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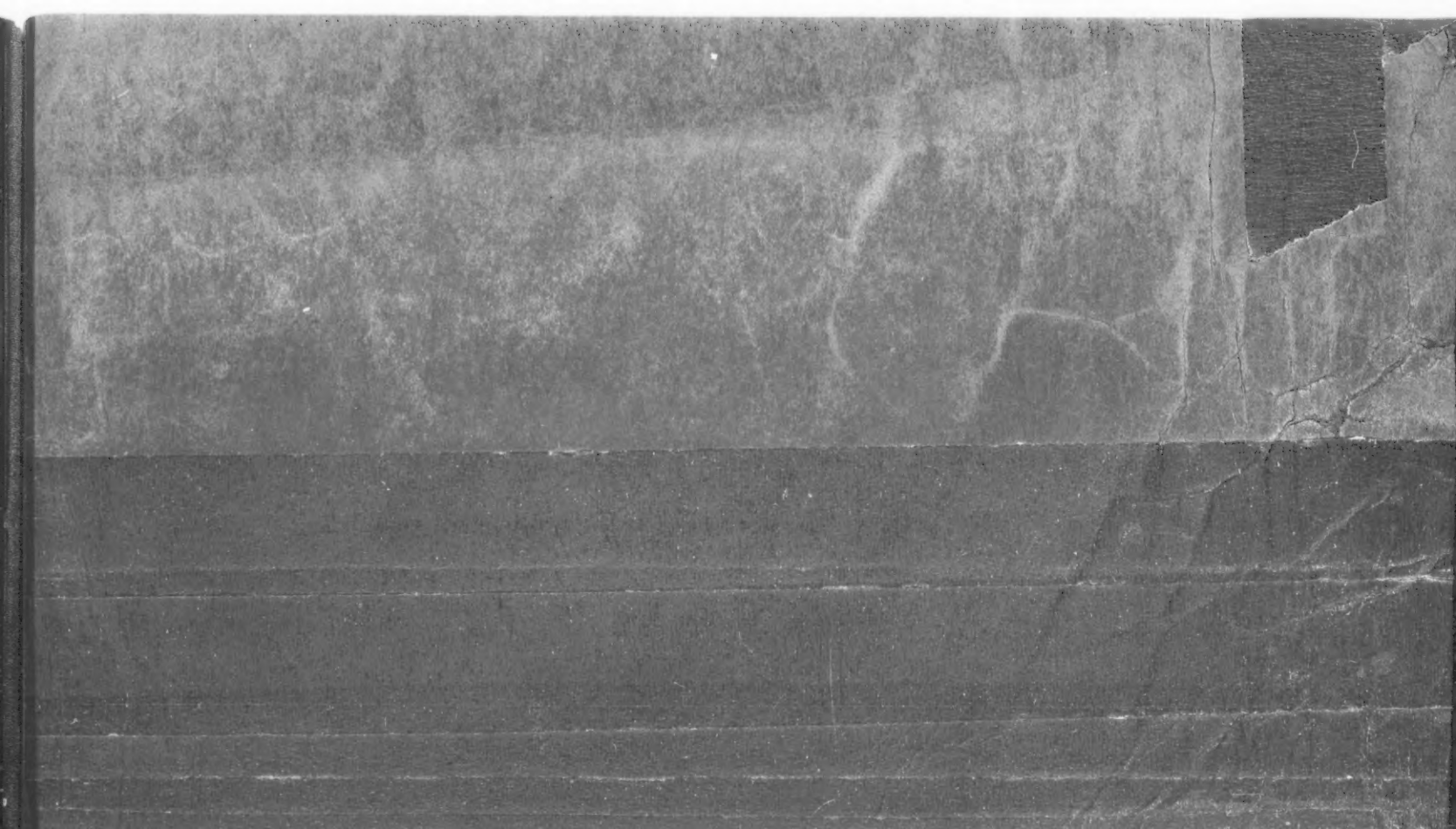

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# HOMEOSTASIS IN A SELECTION EXPERIMENT

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In his stimulating book, Lerner (1954) argues that *Genetic Homeostasis* (the tendency of populations of outbreeding species to resist the effects of artificial selection) arises because selection brings about inferior *Developmental Homeostasis* (canalisation of individual development). He argues, from the frequently observed inferior developmental homeostasis of homozygotes, that in these species good control of development depends on heterozygosity as such. Selection necessarily involves inbreeding and must therefore reduce the heterozygosity of the population and hence produce impaired developmental homeostasis. Natural selection will therefore operate in favour of heterozygosity and resist the artificial selection. In due course the selection pressures will balance and the artificial selection will cease to be effective.

This thesis supposes that the three following hypotheses are generally applicable to artificially selected populations. First, artificial selection must produce deterioration of developmental homeostasis. Second, this poor homeostasis must be the result of decreased heterozygosity. Third, the poor homeostasis must limit significantly the rate of response to artificial selection. The experiment to be reported here bears on the first two of these hypotheses.

## 1. THE MEASURE OF DEVELOPMENTAL HOMEOSTASIS

The kinds of quantity that may be taken as indicating the degree that development is under control have been discussed elsewhere (Thoday, 1953, 1956), and reasons have been given for preferring measures of within-individual variation to measures of between-individual variation. In selection experiments, since their whole success depends upon genetic heterogeneity, between-individual variance can only provide a measure of developmental homeostasis if the proportion of total variance that is genetic can be accurately estimated : a measure of within-individual variation is therefore peculiarly appropriate. The convenient character first studied by Mather (1953*a*), bilateral asymmetry of sternopleural chaeta-number in *Drosophila melanogaster*, has therefore been used in this work.

The choice of this character was based in the first place on the reasonable supposition that in a well-canalised individual the end-results of development on the two sides of the fly are likely to be very similar, whereas in a poorly-canalised individual any tendency

for development of the two sides to diverge will be inadequately buffered (Mather, 1953*a*). Such theoretical considerations alone will not convince everyone, and the author has heard doubts of the value of sternopleural asymmetry as a character, and even doubts of the value of using chaeta number as a character in any experiment in quantitative genetics. These doubts seem to be based on a feeling that chaeta number is a trivial character of little, if any, adaptive significance.

There can, however, be no doubt that chaeta number has adaptive

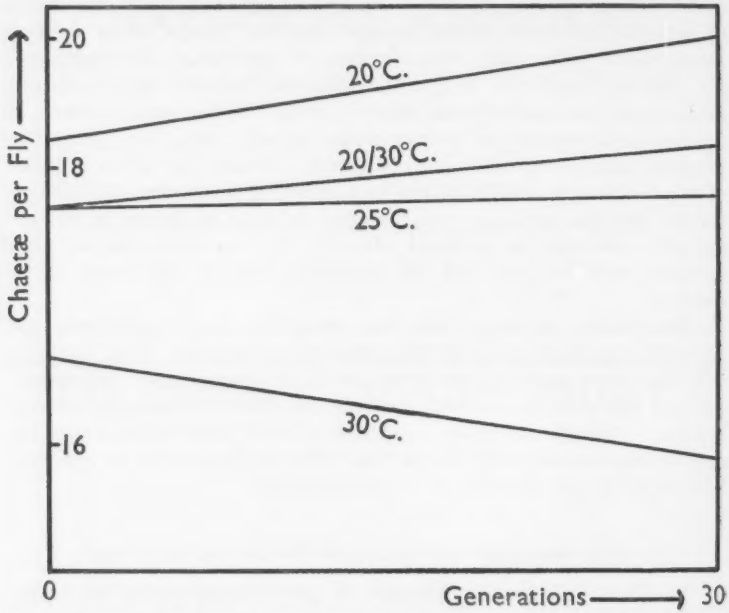


FIG. 1.—The regression lines of sternopleural chaeta number on generations in populations adapting to different environments. Each line is obtained from 3 populations each started from an $F_1$ cross between the inbred lines Oregon and Samarkand. (Log scale). From Beardmore (1956).

significance, for it is affected by *natural* selection. On general grounds alone this is clear : different species of *Drosophila*, and different populations of a species have different characteristic chaeta numbers, and the variance of chaeta number is very much less than the potential genetic variance can be shown to be by appropriate selection experiments (*e.g.* Sismanides, 1942 ; Mather and Harrison, 1949 ; Rasmusson, 1955). These facts must imply a history of effective stabilising selection (Mather, 1953*b*).

The conclusion based on these general considerations is supported by the behaviour of sternopleural chaeta number in populations of *Drosophila melanogaster* studied by Beardmore (1956) who has kindly

permitted me to outline them. Beardmore established three populations in each of four environmental conditions, 20° C., 25° C., 30° C. and an incubator in which the mean temperature is 25° C., but in which there is a smooth diurnal temperature cycle from 20° midnight to 30° C. midday. All the populations were maintained by, as far as possible, a three-weekly transfer of ten pairs of flies and had been started from the same cross between two lines, Oregon and Samarkand, both long inbred at 25°. (Great difficulty was experienced in maintaining the 30° populations, and their incubator was at first left at 28° and only gradually raised to 30°. They have not yet adapted to 30° and, periodically, resort has to be made to insurance cultures kept for one generation at 25° to prevent them dying out.)

TABLE 1

*Significance of changes of chaeta number in adapting populations*

(*From Beardmore, 1956*)

| Environment | Joint regression | Sign of *b* | Differences between three replicate populations | |
|---|---|---|---|---|
| | | | Difference of regressions | Difference of means |
| 20° C. | $t_{(54)} = 3{\cdot}366$ P. $\simeq 0{\cdot}001$ | + | P. large | P. large |
| 25° C. | $t_{(60)} = 2{\cdot}407$ P. $< 0{\cdot}02$ | + | P. $< 0{\cdot}05$ | P. $< 0{\cdot}001$ |
| 30° C.* | $t_{(42)} = 2{\cdot}789$ P. $< 0{\cdot}01$ | − | P. large | P. $\simeq 0{\cdot}01$ |
| F. 20°/30° C. | $t_{(66)} = 2{\cdot}811$ P. $< 0{\cdot}01$ | + | P. large | P. $< 0{\cdot}01$ |

* See footnote to Table 2.

Fig. 1 illustrates the results. The first point of note is that adaptation to the new environments does involve changes of chaeta number. The regressions of chaeta number on generations are significant (table 1), and the slopes are greater in the new environments than in the old. Further, in the old environment the regressions of replicate populations are not consistent, whereas in the new environments they are.

The second point is that the directions of change in 20° and 30° differ, which can only be taken as evidence that it is a good thing for flies developing at 20° to have more chaetæ, and a good thing for those developing at 30° to have less chaetæ. In other words, chaeta number measures something of adaptive significance.

A third point is of the greatest importance. It is well known (Plunkett, 1927) that *Drosophila* cultured at higher temperatures produce fewer chaetæ than those cultured at lower temperatures. This environmental effect is the reason for the differing chaeta numbers of the flies at the start of Beardmore's experiments (fig. 1).

Without Beardmore's results, interpretation of this phenotypic effect of the environment would be difficult. It might on the one hand be held that the environmentally caused modification of chaeta

number arose through poor canalisation of individual development. On the other hand it might be held that the modification of chaeta number arises because the individual is capable of adapting its development so as to produce a phenotype approaching the optimum required in the environment in which development takes place. This is the standard problem, discussed by Thoday (1953), of determining whether inter-individual variation is evidence of good or bad developmental homeostasis. Beardmore's results seem to settle the question for this character, this environmental variable and these flies. The genetic changes in his 20° and 30° populations augment the standard effects of the environment. It follows that the individual fly modifies its development *adaptively* in producing more chaetæ in lower temperatures and fewer chaetæ at higher temperatures.

TABLE 2

*Significance of changes of asymmetry in adapting populations*
*(From Beardmore, 1956)*

| Environment | Joint regression | Sign of *b* | Differences between three replicate populations | |
|---|---|---|---|---|
| | | | Difference of regressions | Difference of means |
| 20° C. | $t_{(84)} = 1{\cdot}887$ P. $<0{\cdot}1$ | − | P. large | P. large |
| 25° C. | $t_{(60)} = 0{\cdot}008$ P. large | + | P. large | P. large |
| 30° C. * | $t_{(42)} = 2{\cdot}530$ P. $<0{\cdot}02$ | − | P. large | P. $<0{\cdot}2>0{\cdot}05$ |
| F. 20°/30° C. | $t_{(66)} = 2{\cdot}989$ P. $<0{\cdot}01$ | − | P. large | P. $\simeq 0{\cdot}05$ |

* The 30° regressions ignore the data obtained before the incubator had been raised to 30° C.

It cannot, of course, be argued from this that higher variance of chaeta number is always to be taken as a measure of higher developmental homeostasis. There is in fact evidence (Thoday, 1956) that high within-culture non-genetic variance is sometimes evidence of low developmental homeostasis (see also Lewontin, 1956), which merely serves to underline the value of measures of within-individual variance such as have been used by Mather (1950, 1953*a*), Jinks and Mather (1955), Paxman (1956), Thoday (1953, 1956), and Tebb and Thoday (1954).

Beardmore's results show that sternopleural number is of adaptive importance, a fact of significance for our argument. If this were not so, it would be less easy to suppose that sternopleural asymmetry might be of importance. His results also provide direct evidence that sternopleural asymmetry is a character of adaptive significance. Table 2 demonstrates the significance of changes in sternopleural asymmetry in his adapting populations. Asymmetry was measured by summing the differences of chaeta number between the two sides of a standard number of flies, a measure differing from that used by

Mather (1953*a*), but the same as that used by Tebb and Thoday (1954). There is no indication of regression on generations in the 25° populations. Each of the other populations (in new environments) shows a negative regression of asymmetry on generation and this is significant in the F. 20/30° and in the 30° populations. The regression in the 20° populations is not quite significant, but that this regression should be the least significant is to be expected, since 20° seems to be as good an environment as 25° for Oregon/Samarkand $F_1$ flies (Thoday, 1953). Furthermore, the rise in chaeta number in the 20° populations might be expected to raise asymmetry as a result of the scaling phenomenon referred to below, and this would mask any improvement of symmetry that was occurring. This same scaling problem somewhat weakens the evidence provided by the 30° populations whose chaeta number decreased. It cannot, however, affect conclusions based on the fluctuating environment populations, so that the overall result is clear : sternopleural asymmetry decreased as the populations adapted to particular new conditions. It must be concluded that natural selection promoted sternopleural symmetry, which, therefore, is of adaptive significance. The same conclusion follows from the experimental results of Tebb and Thoday (1954).

It would in fact be very surprising if bilateral symmetry of chaeta number were not of adaptive significance, since chaeta number must measure some of the attributes of a particular set of differentiation fields, and hence numerical symmetry must be a partial measure of the similarity of the comparable differentiation fields on the two sides of the fly.

A further point arises concerning the relevance of sternopleural asymmetry as a measure of homeostasis. Mather (1953*a*) argued that asymmetry is not so much a result of variation of external environment, but a consequence of internal " accidents of development ". Similar arguments have been used by Reeve and Robertson (1954) concerning abdominal chaetæ and Waddington, Graber and Woolf (1957), who refer to asymmetry as developmental "noise". Our own results (Thoday, 1956 ; Tebb and Thoday, 1954 ; Beardmore, 1956) clearly show that major changes of external environment can affect sternopleural asymmetry, but this does not mean that the divergence of the two sides in development is initiated by a difference of the external environment of the two sides : accidents of development may be more frequent or less well buffered in an unfavourable environment. It is quite probable that we are, in fact, dealing with accidents of development, but this has little, if any, bearing on the validity of sternopleural asymmetry as a measure of the competence of developmental control. Just as a " good " genotype will buffer the individual against undesirable effects of external variables, so a " good " genotype will lead to fewer accidents of development and better buffering of the individual against the effects of such accidents as do occur.

## 2. SCALING PROBLEMS

One disadvantage of sternopleural asymmetry is that it presents problems of scaling when it is desired to use it for the comparison of two populations whose mean sternopleural chaeta numbers differ. Mather (1953*a*) found that there was sometimes no relationship between asymmetry and total sternopleural chaeta number, but that in other types of fly there was a positive relationship. The control lines used in the present experiment show a highly significant positive relationship (calculation by Beardmore, 1956). The experiment involves lines selected for sternopleural chaeta number and some scaling correction is therefore required. The correction used by Thoday (1956), which involves dividing summed asymmetry by total chaeta number, seems to be satisfactory for the population used here since it gives comparable results for high and low selection lines. Asymmetry results are therefore presented as A/T, that is, the summed differences between the two sides of a set of flies (sign ignored) divided by their total chaeta number.

## 3. MATERIAL AND METHODS

The experiments began with a wild stock (" Dronfield ") of *D. melanogaster*, derived from the progeny of a single inseminated female captured near Sheffield. Since its capture the stock had been maintained for six months by standard culture at 25° C., 4 pairs of flies being transferred each generation. This stock was then split into two lines, I and II, and in the next generation 4 single-pair cultures labelled *a*, *b*, *c* and *d* were set up from each. Assay and selection for number of sternopleural chaetæ began with the progeny of these eight pairs.

Assay of any line in any generation involved counting the sternopleural chaetæ on each side of 20 females and 20 males from each of four single-pair cultures except in generation O in which 24 of each sex were counted. In this generation the first four were used to establish control lines. From the remainder, the four with highest chaeta numbers were used to establish high selection lines and the four with lowest chaeta numbers were used to establish low lines.

Thus in generation O the following flies were selected from line I : 4 random females and 4 random males from each culture *a* to *d* ; the 4 highest flies of each sex numbered 1 to 4 from each of the four cultures and the 4 lowest flies likewise. These groups of flies were used to establish lines I C, I H, I L.

Each such line was set up by mating the female $a_1$ to male $b_1$, $a_2$ to $b_2$, $a_3$ to $b_3$, $a_4$ to $b_4$, $b_1$ to $c_1$, $c_1$ to $d_1$ and $d_1$ to $a_1$ and so on, providing 16 single-pair cultures. Culture $a_1 \times b_1$ was labelled $a_1$ and provided this was successful $a_2$, $a_3$ and $a_4$ were discarded. If $a_1$ failed, $a_2$ was used. If $a_1$ and $a_2$ failed, $a_3$ was used, etc. (An additional mass culture (4 random pairs) was also set up to insure against loss of all four single-pair cultures. Resort to this culture was only necessary on four occasions, that is for 4 of the 244 cultures assayed.) The progeny of the successful culture were assayed and selected and the next generation set up as before except that the mating system was changed to $a \times c$, $b \times d$, $c \times a$, $d \times b$. In the following generation the mating was $a \times d$, $b \times a$, $c \times b$, $d \times c$, and in the following the first mating system was repeated. Thus the four single pairs used in each generation are maintained as one population and the labels *a*, *b*, *c* and *d* merely designate female lines.

In the control lines the first 4 flies of each sex that were counted from each culture were labelled 1 to 4 and used. In the selection lines the " best " 4 were used. " Best " was defined as, *e.g.* in a high line, the fly with the most sternopleural chaetæ,

but, if choice were necessary, the most symmetrical fly was used, that is, that with the most nearly equal numbers of chaetæ on its two sides.

In addition to the lines, $F_1$ cultures were also obtained in most generations from crosses between I C and II C, I H and II H, I L and II L and between High and Low lines. Each $F_1$ assay involved 20 flies of each sex from each of 4 single-pair cultures, two of each reciprocal cross. In the H×L $F_1$s the four cultures were of

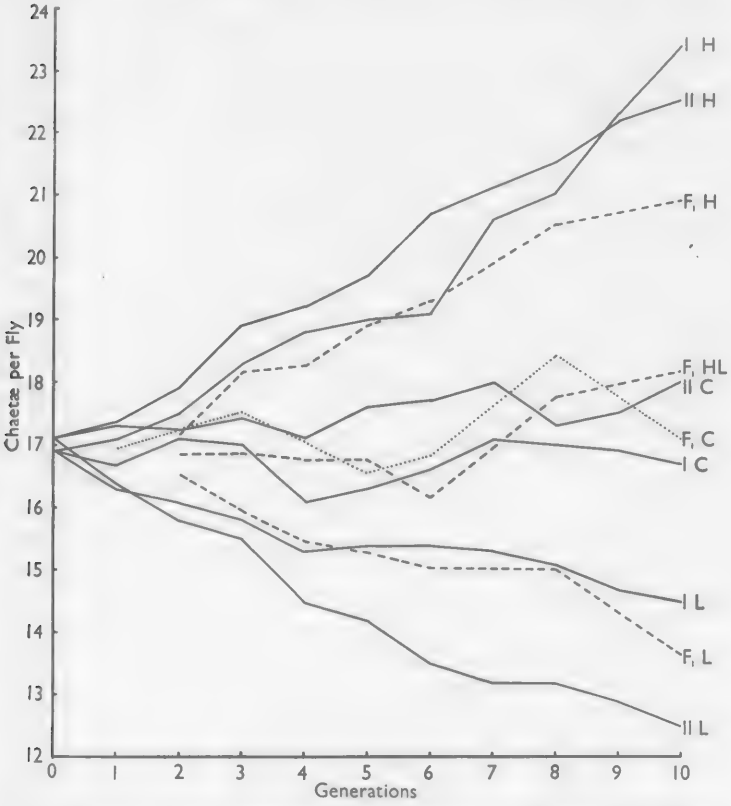


FIG. 2.—Mean sternopleural chaeta number in the lines and their $F_1$ crosses. Solid curves, the lines. Broken curves, the $F_1$s between selected lines. Dotted, the $F_1$s between the control lines. (The $F_1$s are plotted with the generation with which they were cultured.)

4 crosses, I H× II L, II L×I H, II H×I L and I L×II H. Parents for $F_1$ cultures were chosen at random.

All cultures were set up in standard half-pint milk bottles with standard corn-meal black-treacle agar medium, inoculated with live baker's yeast. Counts and matings were made on Tuesdays and Wednesdays, the mating pairs being placed in 2×1 in. tubes. The cultures were set up on Saturday (having been poured on Thursday and yeasted Friday), and the parents were removed on Tuesday. Emergence of adults began the following Monday and the flies were sexed and separated morning and evening until and including Thursday, by which time

emergence was usually complete. The flies were preserved until Tuesday for counting. This routine means keeping the flies for some time and forces a three-week generation, but it is simple to manage and usually permits collection of all the progeny before selection, a matter that may be of some importance (*cf.* Durrant, 1955).

## 4. RESULTS

Fig. 2 shows that the response to selection in the four selection lines was good, and that the control lines, though differing somewhat, remained fairly steadily near their original chaeta numbers.

Table 3 gives the A/T figures, multiplied by 1000 for convenience, for each of the six lines, and for each of the four $F_1$s which are classed with the generations with which they were grown.

TABLE 3

*A/T × 1000 for the lines and their $F_1$ crosses*

| Generation : | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| I Control . . | 52 | 56 | 53 | 46 | 58 | 50 | 53 | 52 | 46 | 50 | 53 |
| II Control . . | 49 | 49 | 55 | 54 | 58 | 62 | 53 | 54 | 56 | 50 | 51 |
| $F_1$ . . . | ... | 50 | 57 | 58 | 48 | 55 | 53 | ... | 54 | ... | 60 |
| I High . . | ... | 47 | 59 | 60 | 51 | 50 | 58 | 62 | 53 | 58 | 59 |
| II High . . | ... | 50 | 54 | 62 | 62 | 54 | 62 | 61 | 63 | 57 | 66 |
| $F_1$ . . . | ... | ... | 50 | 57 | 62 | 58 | 57 | ... | 48 | ... | 56 |
| I Low . . | ... | 55 | 48 | 53 | 56 | 57 | 57 | 54 | 55 | 57 | 64 |
| II Low . . | ... | 55 | 58 | 49 | 57 | 52 | 59 | 52 | 56 | 55 | 62 |
| $F_1$ . . . | ... | ... | 49 | 55 | 53 | 53 | 55 | ... | 58 | ... | 62 |
| High/Low $F_1$ . | ... | ... | 54 | 50 | 56 | 56 | 58 | ... | 55 | ... | 58 |

Fig. 3 shows A/T for the selected and the control lines and table 4 the corresponding analysis of variance. The following points emerge :

1. A/T is higher in the selection lines than in the controls.
2. There is no significant difference between mean A/T for high and low lines, indicating that A/T is a measure satisfactorily compensating for the relationship between arithmetical asymmetry and total chaeta number.
3. A/T does not vary significantly with generation in the controls, but there is a highly significant increase with generations of selection. Furthermore there is no suggestion of differences between the regression slopes of the four selection lines. Both high and low selection result in such an increase. (The regression coefficients of A/T × 1000 on generation are : I C −0·3000, II C 0·1091, I H 0·6545, II H 1·2545, I L 0·8364, II L 0·6091).

The mean bristle numbers for the $F_1$ cultures are illustrated in fig. 2, along with those of the lines. They are plotted with the generation with which they were cultured, not with that which provided their parents. Having due regard to the fact that the parents of the $F_1$ cultures were chosen at random, it seems clear that the bristle numbers of $F_1$ flies show little evidence of overall dominance.

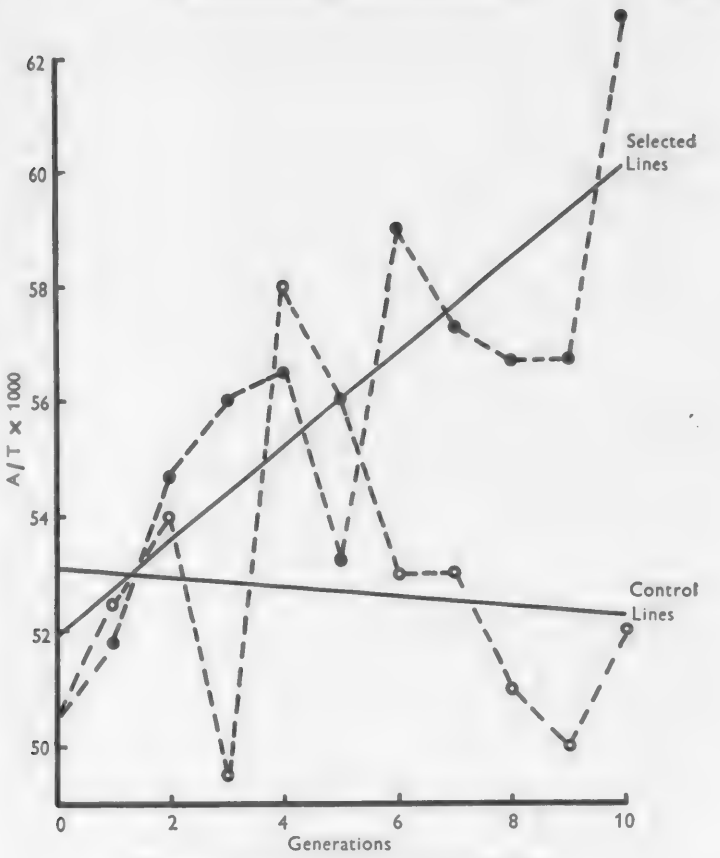


FIG. 9.—Asymmetry in the selected and control lines.

TABLE 4

*Analysis of variance of A/T× 1000 for the lines*

| Source | Sum of squares | N | M.S. | P |
|---|---|---|---|---|
| Joint regression on generations . | 183·4909 | 1 | 183·4909 | ≃0·001 |
| Difference selected and control regressions | 127·9704 | 1 | 127·9704 | <0·01 |
| Difference High and Low regressions | 5·9113 | 1 | 5·9113 | ... |
| Difference selected and control means | 150·6439 | 1 | 150·6439 | <0·01 |
| Difference High and Low means . | 31·0834 | 1 | 31·0834 | ... |
| Residual differences between means | 66·4090 | 3 | 22·1363 | ... |
| Error . . . . . | 866·2638 | 53 | 16·3446 | ... |
| Total . . . . | 1431·7727 | 61 | ... | ... |

Table 3 lists the A/T values for the $F_1$ cultures and those for the $F_1$s between the selected lines are illustrated in fig. 4. Table 5 gives

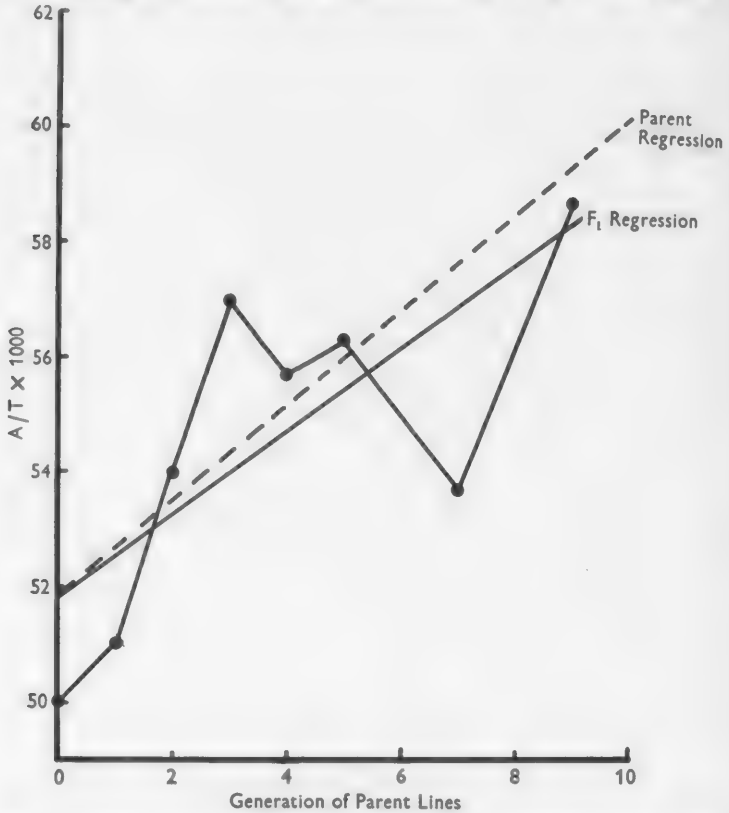


FIG. 4.—Asymmetry of the $F_1$s between selected lines. The regression for the selected lines is plotted as well as the $F_1$ joint regression. (The $F_1$s are plotted with the generation that provided their parents.)

TABLE 5

*Analysis of variance of $F_1$ $A/T \times 1000$. Selected and controls*

| Source | Sum of squares | N | M.S. | P |
|---|---|---|---|---|
| Joint regression on generations . | 120·8989 | 1 | 120·8989 | ≃0·01 |
| Difference between selected and control regressions | 1·8169 | 1 | 1·8169 | ... |
| Residual difference between regressions | 42·0739 | 2 | 21·0369 | ... |
| Error . . . . . | 309·1791 | 24 | 12·8825 | ... |
| Total . . . . | 473·9688 | 28 | ... | ... |

the analysis of variance of $F_1$ A/T. The joint regression of A/T on generation for the $F_1$s is significant and there is no significant difference of behaviour over the generations between the different types of $F_1$. However, the control $F_1$ regression is not by itself significant and it is lower than the joint regression for the selected $F_1$s. The control $F_1$s do not therefore provide sufficient data to be useful. The $F_1$s between the selected lines, on the other hand, do. It is clear (fig. 4) that the $F_1$s between the selected lines increase in A/T in exactly the same way as the selected lines themselves.

Now if the deterioration of the selected lines arose because of increasing homozygosity, their $F_1$s should show lower A/T values than the lines, unless the lines crossed to give the $F_1$s had identical genotypes, which is unlikely for all and impossible for the H $\times$L $F_1$s. It would therefore appear that increasing homozygosity in the selected lines cannot be invoked as an explanation of their increasing asymmetry.

There is, however, one possibility that might render this conclusion invalid. If asymmetry were a character *entirely* determined by maternal genotype and completely independent of the genotype of the individual in which it is measured, then the $F_1$s would be expected to show the same asymmetry as the parent. If this asymmetry arose from maternal homozygosity. we should then expect reduced asymmetry in $F_2$, for the $F_2$ would have heterozygous mothers. Mather (1953*a*) did in fact find some evidence that asymmetry might *in part* be determined by maternal genotype, but only *complete* maternal inheritance would invalidate the conclusion reached above. Further, 25 $F_2$ cultures raised from the High $\times$Low $F_1$ after the completion of the experiment gave A/T values identical with those of 16 contemporaneously reared $F_1$ cultures (A/T $\times$1000 $F_1$ 56·3, $F_2$ 56·0), and it seems clear that maternal inheritance cannot be important in this experiment. It must be concluded that homozygosity is not an important factor determining the observed increase of asymmetry.

## 5. DISCUSSION

The results presented show clearly that directional selection can cause deterioration of the systems responsible for developmental homeostasis as here measured, and to this extent they are in agreement with Lerner's thesis. They do not, however, lend any support to Lerner's hypothesis that increasing homozygosity is the cause of the deterioration.

There are two alternative explanations which may account for the increased asymmetry. The first is similar to that put forward by Mather to explain declining fertility in selection lines. Selection produces unbalance in gene complexes linked to the genes directly concerned with the character under selection and the result is impaired fertility (Mather and Harrison, 1949). The increased asymmetry may merely be another manifestation of this poor balance.

In a discussion of the results obtained part way through the present experiment, and of those obtained with some other lines, the author (Thoday, 1956), put forward a second explanation similar to that discussed by Falconer and Robertson (1956). This was that, in selecting for a given character, we may pick out from a population not only individuals whose genotypes determine directly an extreme value for that character, but also individuals whose genotypes render them exceptionally subject to modification by the environmental variation in the culture, or exceptionally incompetent at buffering the effects of internal accidents of development, or exceptionally subject to such accidents. Selection of these latter types would lead to increase of genotype-environment interaction and/or deterioration of developmental stability. That such genes exist in *Nicotiana* has been made abundantly clear by the work of Mather and his associates (Jinks and Mather, 1955; Paxman, 1956), and in addition Mather (1953*a*) has demonstrated that sternopleural asymmetry in *Drosophila* is itself a character responsive to artificial selection. Such genes would be selected in this experiment. Provided that the same genes, or some of the same genes, that affect inter-individual variance of sternopleural number also affect asymmetry in the same direction, the present increase of A/T in the selection lines is formally explicable on the basis of such selection.

Evidence concerning coefficients of variation in the lines of this experiment might be expected to provide relevant information. Unless new mutants arise or fixed variability becomes freed at a greater rate than the available genetic variance is used up, genetic variance must decline in lines undergoing inbreeding, such as the control lines in this experiment, and in selection lines. Phenotypic variance must therefore decline unless genotype-environment interaction and/or developmental variation caused by intrinsic accidents during development increase. Selection for increase of these will, on the other hand, arrest the decline and may even produce an increase of phenotypic variance. The final result will depend upon the relative variance arising from stability genes and additive genes at the beginning of selection.

In these experiments, the variance of the control lines behaved as expected. Some of the selection lines, on the other hand, increased in variance. Furthermore, the regression slopes for coefficients of variation for the different lines fall in the same order as those for A/T, which supports the view that A/T and coefficient of variation have some common causes. The changes in variance are therefore in agreement with the hypothesis that the decline of developmental homeostasis observed in the selection lines results from the selection for low developmental stability, which in small or large degree is a necessary consequence of directional selection. This evidence is, however, of no real value, for the changes of variance are not significant and are open to other interpretations. The alternative balance

hypothesis would explain them and the freeing of fixed variability in the selection lines would also explain them.

Distinction between the balance hypothesis, and that involving direct selection of genes for poor homeostasis is, however, possible. If the deterioration of homeostasis arises as a result of the selection of unbalanced gene-complexes linked to chaeta number genes, then increase of asymmetry should be greatest when the selection of extreme chaeta number genotypes is most effective. If, on the other hand, increase of asymmetry arises because of the selection of individuals whose extreme chaeta-number is caused by poor developmental canalisation instead of an extreme chaeta-number genotype, then

TABLE 6

*Correlation between increase of asymmetry and* $h^2 = \Delta P/i$ *in the selected lines*

| Generation | 0-1 | 1-2 | 2-3 | 3-4 | 4-5 | 5-6 | 6-7 | 7-8 | 8-9 | 9-10 |
|---|---|---|---|---|---|---|---|---|---|---|
| A/T increase . | −4·9 | 11·7 | 1·0 | −8·5 | −0·5 | 7·3 | 4·0 | −8·6 | 4·5 | 1·5 |
| I H $h^2$ . . . | 0·05 | 0·19 | 0·30 | 0·17 | 0·05 | 0·04 | 0·39 | 0·13 | 0·33 | 0·27 |
| A/T increase . | 1·4 | 4·3 | 7·6 | 0·4 | −8·2 | 8·2 | −1·1 | 1·4 | −5·7 | 8·8 |
| II H $h^2$ . . . | 0·06 | 0·17 | 0·26 | 0·09 | 0·19 | 0·30 | 0·14 | 0·08 | 0·21 | 0·07 |
| A/T increase . | 3·1 | 0·8 | 4·8 | 3·4 | −8·5 | 9·2 | −2·7 | 1·2 | 2·0 | 7·1 |
| I L $h^2$ . . . | 0·33 | 0·07 | 0·06 | 0·10 | −0·26 | 0·00 | 0·06 | 0·13 | 0·20 | 0·08 |
| A/T increase . | 6·3 | 7·5 | −8·2 | 8·0 | −4·8 | 6·6 | −6·3 | −3·5 | −0·7 | 6·6 |
| II L $h^2$ . . . | 0·32 | 0·27 | 0·15 | 0·44 | 0·14 | 0·32 | 0·13 | 0·26 | 0·19 | 0·37 |

increase of asymmetry should be greatest when the selection of extreme chaeta-number genotypes is least effective.

Accordingly the regression of increase of asymmetry in a generation on the advance of chaeta number made in that generation, measured by the heritability formula $h^2 = \Delta P/i$ (Lerner, 1950), has been calculated (table 6).

The regression is positive and just significant ($t_{(38)} = 2{\cdot}22$, $P < 0{\cdot}05$) suggesting that the balance hypothesis is the more important, and providing no evidence that the other effect is operative. It should, however, be borne in mind that the two hypotheses are not mutually exclusive and that direct selection of instability genes may also be effective. It might even be more important than the balance effect if heritability were relatively low and response of the primary character to selection were small, as was suggested in explanation of the behaviour of the *vg* line described by Thoday (1956).

We may now suggest the following history for poor homeostasis in a longer term selection experiment. If, at the start of the experiment, the available genetic variance directly affecting the primary character is relatively high, selection will be effective in changing the mean, but development will become gradually less stable as linked gene complexes affecting stability become unbalanced. The resulting

decrease in heritability of the primary character may render more likely the direct selection of instability genes (as might occur if heritability were low at the beginning). Stability should therefore deteriorate at an increasing rate except in so far as the genetic variance of stability is used up. If, however, after a period of selection, new genetic variance directly affecting the primary character were to be produced in the line, either by mutation, recombination, or gene-interactions increasing the expressivity of existing heterozygous loci, then a new balance of selection pressures would be set that might permit rapid improvement of stability. Accelerated response to selection, or the crossing of selection lines might therefore, on these grounds, result in improvement of stability. Such was the behaviour of the *dp* line described by Thoday (1956), which showed a clear improvement in sternopleural symmetry at the same time as an accelerated response to selection. After such a response we would expect stability to begin to deteriorate once more. Whether or no the final level of instability reached might prove an effective bar to full exploitation of the genetic variance directly affecting the primary character would depend on the degree of correlation of stability with fitness in the particular population and environment. If it were an effective bar, coincident artificial selection, both for the primary character and for stability, might in the long run produce more satisfactory results than selection for the primary character alone.

## 6. SUMMARY

(1) It is argued from results of Beardmore that asymmetry of sternopleural chaeta number is a useful measure of developmental homeostasis.

(2) Ten generations of selection for high or low sternopleural chaeta number produced a deterioration of developmental homeostasis so measured.

(3) This decline of developmental homeostasis was not caused by increasing homozygosity, for the same deterioration was observed in crosses between the selected lines.

(4) Two alternative hypotheses are suggested. One involves direct selection of " poor-homeostasis " genes. The other involves the deteriorating balance of gene complexes linked to those directly affecting bristle number. The results indicate that the second of these is correct, but it is suggested that direct selection of instability genes may be more important when heritability is relatively low, the balance effect being more important when heritability is high enough to permit good response to selection.

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# SPLIT PETALS IN CRUCIFERS

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## 1. INTRODUCTION

FLOWERS of most Crucifers have four entire petals, six stamens and a two-valved ovary. In members of the genera, *Matthiola*, *Raphanus* and *Brassica* lines or plants have been found in which some flowers deviate from this formula. In *B. napus* (swede), *B. oleracea* and *R. sativa* (radish) some of the petals were divided near their base, and lines in which this was fairly common were isolated. This division probably resulted from an early splitting of the petal primordia. Plants with the same abnormality have been seen in *M. bicornis*.

TABLE 1

*Number of normal and abnormal flowers at different times*

| Plant | 10/10/55 | | 13/10/55 | | 17/10/55 | | 20/10/55 | | 25/10/55 | | 28/10/55 | | 1/11/55 | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | N. | A. | N. | A. | N. | A. | N. | A. | N. | A. | N. | A. | N. | A. |
| 9052/1 | 15 | | 30 | 4 | 57 | 5 | 70 | 2 | 48 | 20 | 80 | 48 | 70 | 60 |
| 9052/4 | 5 | | 25 | 2 | 46 | ■ | 70 | | 100 | 7 | 120 | 12 | 150 | 20 |
| 9053/2 | 8 | 1 | 32 | | 71 | 1 | 90 | 9 | 70 | 44 | 80 | 60 | 120 | 30 |
| 9054/2 | 1 | | 18 | | 33 | | 64 | | 76 | ■ | 100 | 6 | 120 | 17 |
| 9054/4 | 12 | | 33 | | 58 | 1 | 60 | | 100 | 2 | 110 | 16 | 150 | 5 |
| 9054/5 | 18 | | 42 | | 66 | 1 | 50 | 7 | 99 | 21 | 100 | 53 | 160 | 20 |

In the radish population which produced divided petals, some stamens were reduced or absent, or fused with petals or the pistil, and some petals reduced or absent.

In *B. napus*, *B. oleracea* and *M. bicornis* usually only one petal on a flower was affected, though as many as three have been noted. In affected radish flowers, divided petals were often more numerous.

It is not known whether the stamen irregularities in the radish were causally connected with the petal abnormality, or whether a separate agent was responsible for their appearance. The frequency of affected flowers on different swede plants has varied from $\frac{1}{2000}$ to $\frac{1}{2}$. In both the swede and radish, divided petals were not distributed on the plants in any clear consistent pattern. However, their appearance has not been at random, as their frequency varied during the flowering season (see table 1).

On the swede, numerous branches flower at the same time, but there was no conclusive evidence that the proportion of affected flowers varied from branch to branch at any one time. Of 64 between-branch comparisons tested in 1953, $\chi^2$ was significant at the 5 per cent. level in three cases, and at the 1 per cent. level in two.

In the radish, affected flowers on plants with a large number of abnormal flowers had more divided petals than on plants with a smaller proportion of affected flowers.

Some results on the inheritance of this character are reported here.

## 2. MATERIAL

The inheritance of this abnormality has been studied in the following populations.

1. Families of Calder swede.
2. Families of a radish population known locally as Chinese turnip. Divided petals were common on many plants in both these populations.
3. $F_2$'s between Calder and some unrelated normal swedes.
4. $F_1$, $F_2$, $F_3$, parental and back-cross generations of hybrids between an abnormal Calder swede plant and a normal Grandmaster plant. No divided petals have been seen on some 15,000 Grandmaster flowers.

Lot 1 was grown in 1951, 1953 and 1954; lot 2 in 1951 and 1953; lot 3 in 1955; and lot 4 in 1954, 1955 and 1956.

In 1951 and 1953, seeds were sown in their permanent positions in the open. In 1954 seeds were sown in the autumn and transferred to the glasshouse in the spring. In 1955 some were planted in the glasshouse and some in the field. The glasshouse consisted of several cells, and each plant was assigned to a cell at random.

Flowers were classified every three or four days. With this interval, no flower was scored twice but few were missed.

Throughout, N stands for a normal flower, or for a plant with no abnormal flowers, A for an abnormal flower or for a plant with any abnormal flowers.

## 3. VARIATION WITHIN CALDER SWEDE AND RADISH

### (1) *Presence of abnormal flowers*

*Swede 1953.* The families grown were derived from open pollinated Calder plants by two generations of selfing, or were $F_2$'s of crosses between open pollinated Calder plants. Plants with any abnormal flowers were classified as abnormal. Results are shown in table 2.

Apart from the aberrant $F_2$ family in the cross of $6/10 \times 6/9$, these results could be explained by assuming that one dominant gene in this population caused the occurrence of abnormal plants.

*Swede 1954.* Thirteen plants were progeny tested in 1954. Eleven of these were from segregating families, and two were from normal plants. Results are shown in table 3.

The two normal plants tested failed to breed true, and in their progeny only one normal occurred in a total of twelve. Among the progenies of the abnormal plants from mixed families, of which one-third would be expected to be true breeding abnormals, and two-thirds to segregate 1 : 3, only one contained any normal plants, and it only one in a total of 22. Clearly, any idea of a single dominant gene as a cause of the presence of abnormal flowers on plants in the Calder population is not tenable.

*Radish 1953.* In 1951 four plants were inter-crossed and selfed. Plants 1 and 4 were classified as normal, 2 and 3 as abnormal, but

only a few flowers on each plant were examined. Results from these matings are given in table 4.

TABLE 2

*Abnormal plants in Calder families 1953*

| Parent | | | Progeny | | |
|---|---|---|---|---|---|
| Plant no. | Flowers N. | A. | Generation | Plants N. | A. |
| 6/10 | 19 | 1 | $S_2$ | 5 | 7 |
| 6/12 | 1 | 18 | $S_2$ | 1 | 12 |
| 6/3 | 16 | 0 | $S_2$ | 3 | 0 |
| 6/5 | 8 | 4 | $S_2$ | 0 | 7 |
| | | | | 9 | 26 |
| 6/1×6/12 | 6 : 4×1 : 18 | | $F_2$ | 0 | 3 |
| | | | | 0 | 2 |
| | | | | 1 | 3 |
| | | | | 1 | 8 |
| 6/3×6/1 | 16 : 0×6 : 4 | | $F_2$ | 3 | 3 |
| | | | | 1 | 4 |
| | | | | 1 | 9 |
| | | | | 4 | 10 |
| | | | | 9 | 26 |
| 6/10×6/9 | 19 : 1×5 : 2 | | $F_2$ | 1 | 11 |
| | | | | 7 | 2 |
| | | | | 8 | 13 |
| All plants | | | | 27 | 73 |

TABLE 3

*Affected plants in Calder families 1954*

| Parent plant | Abnormal from abnormal family | | Abnormal from mixed family | | Normal from mixed family | |
|---|---|---|---|---|---|---|
| Progeny | N. | A. | N. | A. | N. | A. |
| | 0 | 5 | 0 | 6 | 1 | 6 |
| | 1 | 6 | 0 | 10 | 0 | 5 |
| | 0 | 3 | 0 | 2 | | |
| | | | 0 | 6 | | |
| | | | 0 | 2 | | |
| | | | 1 | 21 | | |
| | | | 0 | 21 | | |
| | | | 0 | 8 | | |
| Total . . | 1 | 14 | 1 | 76 | 1 | 11 |

Some abnormals occurred in the N×N matings, and one normal in the A×A matings. A single gene is not sufficient to account for these results. The A×A matings left more abnormals than the N×A combinations, which in turn gave a higher frequency than the N×N.

### (ii) *Expression of abnormality*

The proportion of abnormal flowers on affected plants varied from plant to plant and from time to time on any one plant. This variation from time to time poses a sampling problem which was not satisfactorily resolved. The percentage of abnormal flowers per plant was computed, and its distribution among plants of these populations was found to be strongly skewed. The distribution of log of the percentage, or of the values obtained by the arc sine transformation, failed to remove

TABLE 4

*Abnormal plants in Radish families*

| Parent | | Progeny | |
|---|---|---|---|
| Type | Plant no. | N. | A. |
| | ♀ ♂ | | |
| N×N | 1 × 1 | 4 | 0 |
| | 4 × 1 | 29 | 2 |
| | 1 × 4 | 3 | 1 |
| | 4 × 4 | 3 | 3 |
| | | 39 | 6 |
| N×A | 2 × 1 | 2 | 5 |
| | 3 × 1 | 5 | 24 |
| | 4 × 2 | 6 | 9 |
| | 2 × 4 | 7 | 8 |
| | 3 × 4 | 4 | 2 |
| | | 24 | 48 |
| A×A | 2 × 2 | 1 | 10 |
| | 3 × 2 | 0 | 19 |
| | 2 × 3 | 0 | 15 |
| | | 1 | 44 |

all this skewness. The second of these transformations has been used in computing means and variances.

Plants with no abnormal flowers also pose a problem. In some cases it is reasonable to assume that the incidence in these plants is low and that they would have produced abnormal flowers had they produced enough flowers. But it is also possible that they represent a different sort of plant which does not produce abnormals. When family means have been computed, all normal plants have been omitted. In fact, there was a correlation between the proportion of affected plants in a family and the percentage of abnormal flowers on a plant, so that when family means are compared, the omission of normal plants tends to reduce family differences.

*Swede 1953.* Progeny means of affected plants for progenies with five or more abnormal plants are shown in table 5. Differences

between families were significant. If family 3449 was omitted, however, the remaining family means were not significantly different.

TABLE 5

*Swede family means 1953*

| Family no. | N. | A. | Mean | Analysis of variance | DF. | M.Sq. |
|---|---|---|---|---|---|---|
| 3443 | 1 | [illegible] | 6·64 | | | |
| 3444 | 4 | 10 | 4·70 | Total . . . . | 45 | |
| 3446 | 1 | 11 | 8·06 | | | |
| 3448 | 1 | 6 | 10·01 | Between families . . | 4 | 274·3 |
| 3449 | 1 | 10 | 18·16 | Within families . . | 41 | 47·3 |

TABLE 6

*Swede family means, 1954*

| Parent | | | Progeny | | | Analysis of variance | | |
|---|---|---|---|---|---|---|---|---|
| No. | Flowers | | No. | Plants | Mean | | | |
| | N. | A. | | N. A. | | | DF. | M.Sq. |
| 3444/2 | 336 | 1 | 5211 | 1 21 | 7·42 | | | |
| 3446/8 | 554 | 47 | 5215 | [illegible] 21 | 8·46 | Total . . . | 72 | |
| 3446/11 | 471 | [illegible] | 5219 | 0 [illegible] | 3·92 | | | |
| 3447/3 | 236 | [illegible] | 5220 | 1 [illegible] | 2·77 | Between families . | 5 | 1277 |
| 3449/7 | 282 | 79 | 5233 | 0 6 | 24·44 | Within families . | 67 | 63 |
| 3449/8 | 307 | 112 | 5230 | 1 11 | 30·10 | | | |

TABLE 7

*Mean abnormal flowers per plant in Radish families, 1953*

| Parents | | Progeny | | |
|---|---|---|---|---|
| Plants | Type | Mean | N. | A. |
| 2×2 | A×A | 26·3 | 1 | 10 |
| 2×3 | A×A | 31·6 | 0 | 15 |
| 3×2 | A×A | 33·3 | 0 | 19 |
| 3×1 | A×N | 30·1 | 5 | 24 |
| 1×2 | N×A | 8·6 | 2 | 5 |
| 2×4 | A×N | 11·3 | 7 | 8 |
| 4×2 | N×A | 13·3 | 6 | 9 |

*Swede 1954.* The relevant data are shown in table 6.

Progeny means were again different. When families 5230 and 5233 were omitted, there were significant differences between the other families. The family with the highest mean in 1953 (3449) left two progenies with the highest means in 1954.

The mean number of flowers classified on each plant was about the same in both years (557 and 547).

*Radish 1953.* Results are shown in table 7.

The families fell into two groups, significantly different from each other, but with no significant differences within each group. The progenies with a high mean had a higher proportion of abnormal plants than those with a low mean.

In the radish, affected flowers often had more than one divided petal. As many as five extra lobes have been recorded, and many flowers had two or three. The mean number of extra petal lobes per abnormal flower was closely correlated with the percentage of abnormal flowers per plant ($r = +0{\cdot}86$).

### (iii) *Discussion*

In both the swede and radish the proportion of abnormal flowers varied from plant to plant and from time to time. In both, differences between family means occurred, and in neither population could the presence of abnormal flowers be explained by the action of one gene. These similarities suggest that genetic control of this character is similar in the two populations. Perhaps the most plausible explanation of these results is that all, or most, plants in these populations are potentially capable of producing abnormal flowers, but that some are more likely to do so than others, this probability being genetically and environmentally modified. In support of this interpretation, several plants with normal phenotypes have produced abnormal progenies, and none which have been well tested have failed to do so. Evidence of genetic modification is provided by the famly differences in mean expression on abnormal plants, and parent-offspring correlations in mean expression, and by the different frequencies of abnormal plants in different families. Differences on the same plant during the flowering season suggest that environmental variations affect character expression.

We shall see in the next section that the potentiality to produce abnormal flowers is not only genetically modified, but also genetically determined.

## 4. VARIATION IN CROSSES BETWEEN CALDER AND GRANDMASTER SWEDES

### (i) *Material*

Divided petals have been observed in only two varieties of swedes, Calder and Crimson King. They have not been seen in Grandmaster though some 15,000 flowers have been examined. Grandmaster was chosen as a normal parent ($P_2$), and crosses were made between it and an abnormal Calder plant ($P_1$). The pedigrees of these plants are listed in table 8.

One plant each of Calder (5201/1), Grandmaster (5242/6) and $F_1$ (5244/6) was used to produce the $F_1$, $F_2$, $P_1$, $P_2$ and back-cross generations. The $F_1$ (5244/6) which had 623 normal and no abnormal flowers, was produced by crossing 3423/2 × 3411/4. In this $F_1$ family there were 16 abnormal and 3 normal plants.

In addition to this set of hybrids, $F_1$'s of some crosses between Calder plants and some unrelated normal swedes, N.H 504 and Y, were grown. These $F_1$'s contained 12 normal plants and 23 abnormals.

### (ii) *Results* 1955

There were no differences between reciprocals of the same cross, and they have been lumped in the following tables and discussion.

The frequency distributions of mean expression in the parents and hybrids is shown in fig. 1.

The $P_1$ distribution appears to be unimodal and continuous, and the $F_2$ and $BC_1$ populations appear to consist of two classes of plants, some producing abnormal flowers, some not, the mean percentage of abnormal flowers being apparently continuously distributed among the affected plants.

It seems reasonable to assume that the ability to produce abnormal flowers is segregating in these populations. The numbers of normal

TABLE 8

*Pedigrees of parent plants*

| Year | Calder parent | | | Grandmaster parent | | |
|---|---|---|---|---|---|---|
| | No. | Flowers N. A. | Sister plants N. A. | No. | Flowers N. A. | Sister plants N. A. |
| 1951 | 6/5 | 8 4 | ... | ... | ... | ... |
| 1952 | 35/6 | 10 0 | 6 1 | ... | ... | ... |
| 1953 | 3423/2 | 72 47 | 0 3 | 3411/4 | ... | ... |
| 1954 | 5201/1 | 752 144 | 0 5 | 5242/6 | 486 0 | 14 0 |
| 1955 | Plots 9051-56 | ... | 2 52 | Plot 9071 | ... | 7 0 |
| 1956 | Plots 1207, 1208 | ... | 5 10 | Plots 1220, 1221 | ... | 16 0 |

*Note.*—Plots 9051-56 and 1207, 1208 were derived by successive selfings from plant 6/5 and plots 9071 and 1220, 1221 from 3411/4, also by selfing.

and abnormal plants in the various hybrid and parent populations are given in table 9.

If the abnormality is caused by one dominant gene with almost complete penetrance in the homozygote, and with penetrance in the heterozygote equal to that in the $F_1$, one would expect the segregation ratios in $BC_1$, $BC_2$ and $F_2$ to be those shown in columns 4 and 5 of the table. As can be seen, the observed and expected ratios are in close agreement. In addition, if one assumes that the mean expression of affected heterozygotes is that of the $F_1$, one would expect the means of affected plants in the $BC_1$, $BC_2$ and $F_2$ populations to be those given in the last column of the table. The observed means fall in the expected order.

The hypothesis of a single dominant gene, with penetrance in the homozygote and heterozygote equal to that in $P_1$ and $F_1$, satisfactorily accounts for the observed data.

Some of these plants were progeny tested in 1956. Data are given in table 10.

The normal $P_1$ (Calder) plant which was tested gave abnormal progeny. So did one of the normal $F_1$ plants, and three of the $F_2$'s.

Normal $P_1$ and $F_1$ plants gave the same proportion of abnormal progenies as their abnormal sisters. If again the proportion of normals

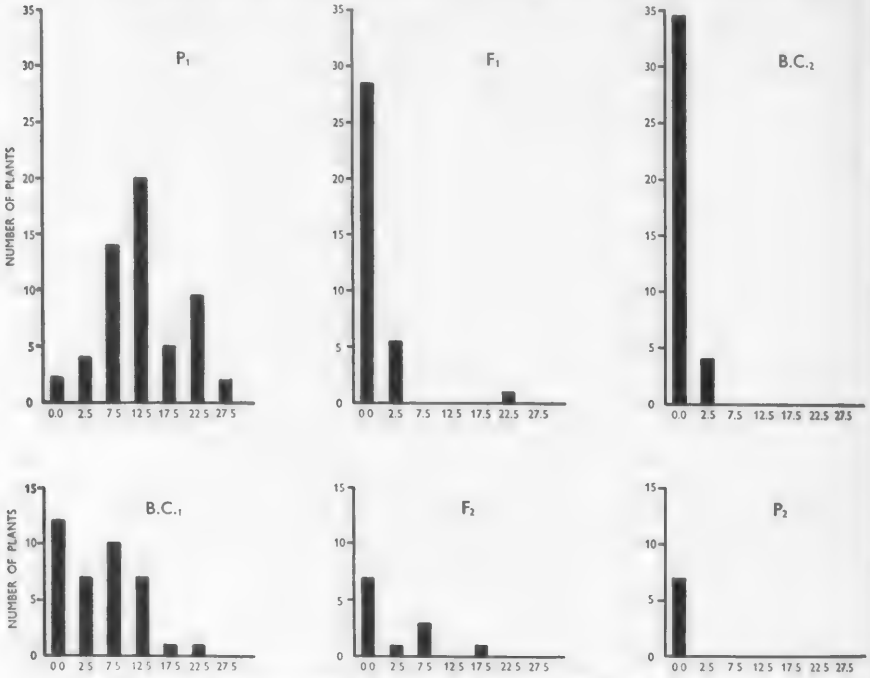


FIG. 1.—Distribution of percentage of abnormal flowers per plant (arc sine transformation) in hybrid swede populations.

TABLE 9

*Normal and abnormal plants in swede hybrids, 1955*

| Generation | N. A. Observed | N. A. Expected | Means of affected plants | |
|---|---|---|---|---|
| | | | Observed | Expected * |
| $P_1$ | 2 52 | ... | 12·41 | ... |
| $P_2$ | 7 0 | ... | ... | ... |
| $F_1$ | 28 9 | ... | 5·18 | ... |
| $BC_1$ | 12 26 | 14·4 23·6 | 9·02 | 11·72 |
| $BC_2$ | 31 4 | 30·9 4·1 | 2·63 | 5·18 |
| $F_2$ | 7 5 | 7·5 4·5 | 7·96 | 10·00 |

* $BC_1 = (F_1+4P_1)/5$ $F_2 = (F_1+2P_1)/3$ $BC_2 = F_1$.

and abnormals in the $P_1$ and $F_1$ is taken as the penetrance ratio in homozygotes and heterozygotes, one would expect the ratio in $F_2$ to be that given in the last column of the table, and an $F_2$ produced

from a random sample of $F_2$ plants would segregate in the ratio also given in the last column. As can be seen, there was again a close fit between observed and expected.

TABLE 10

*Normal and abnormal plants in swede hybrids, 1956*

| Generation | Parent plant | | Progeny plants | | Generation totals | | | |
|---|---|---|---|---|---|---|---|---|
| | No. | Flowers | | | Observed | | Expected | |
| | | N. A. | N. | A. | N. | A. | N. | A. |
| $P_1$ | 9052/2 | 679 0 | 2 | 4 | | | | |
| | 9052/6 | 797 53 | 3 | 6 | 5 | 10 | | |
| $F_1$ | 9051/2×9071/6 | 966 : 164×1085 : 0 | 10 | 0 | 10 | 0 | | |
| $F_2$ | 9057/1 | 1374 ■ | 12 | 0 | | | | |
| | 9057/2 | 1138 0 | 8 | 2 | | | | |
| | 9057/3 | 810 0 | 10 | 0 | | | | |
| | 9068/2 | 1734 1 | 6 | 3 | 36 | 5 | 34·2 | 6·8 |
| $F_3$ | 9069/1 | 914 13 | 4 | 5 | | | | |
| | 9069/2 | 171 20 | 8 | 2 | | | | |
| | 9069/12 | 530 0 | 9 | 1 | | | | |
| | 9069/13 | 272 ■ | 3 | 0 | | | | |
| | 9070/2 | 1039 0 | 8 | 2 | | | | |
| | 9070/3 | 928 0 | 7 | 2 | 39 | 12 | 38·25 | 12·75 |
| $P_2$ | 9071/2 | 820 0 | 10 | 0 | | | | |
| | 9071/3 | 810 0 | 6 | 0 | 16 | 0 | | |

TABLE 11

*Classification of $F_2$ families*

| Plant | Progeny | | Segregation types | | |
|---|---|---|---|---|---|
| | N. | A. | 1 : 0 | 5 : 1 | 1 : 2 |
| 9069/1 | 4 | 5 | * | * | ... |
| 9069/2 | 8 | 2 | * | ... | * |
| 9069/12 | 9 | 1 | * | ... | * |
| 9069/13 | 3 | 0 | ... | ... | ... |
| 9070/2 | 8 | 2 | * | ... | * |
| 9070/3 | 7 | 2 | * | ... | * |

One would expect the $F_3$ families to be of three types, namely all normals, or five normals to one abnormal ($= F_2$), or 1 normal to 2 abnormals ($= P_1$) these types of families occurring in the familiar 1 : 2 : 1 ratio. The six $F_3$ families are listed in table 11.

The asterisks in the cells of the table indicate segregation types to which these families are unlikely to belong. One family (from 9069/13) is too small to furnish any evidence. Of the remainder, one could be from a homozygote, and the other four from heterozygotes. The proportions of normal and abnormal plants in these families is not significantly different from $F_2$.

The other notable feature of these data is the lower rate of penetrance in both homozygotes and heterozygotes in 1956 than in 1955.

In addition to these progeny tests, the $F_1$, $F_2$, $BC_1$, and $BC_2$ generations grown in 1955 were grown again in 1956 from seed reserves. Results from this material are set out in table 12.

TABLE 12

*Repeat of 1955 test*

| Generation | Parents | 1955 | | 1956 | |
|---|---|---|---|---|---|
| | | N. | A. | N. | A. |
| $F_1$ | 5242/6×5201/1 | 28 | 9 | 11 | 3 |
| $F_2$ | 5244/6 S. | 7 | 5 | 12 | 8 |
| $BC_1$ | 5244/6×5201/1 | 12 | 26 | 0 | 20** |
| $BC_2$ | 5244/6×5242/6 | 31 | 4 | 16 | 3 |

Except for the $BC_1$, the tests in the two years are in substantial agreement, and the data from the two years pooled fit the hypothesis. The chances that the two $BC_1$ samples could have come from the same population are somewhere between 1/50 and 1/100, and the results from 1956 do not fit the hypothesis. This discrepancy remains unexplained.

Apart from this, these results, with substantial agreement between the same populations grown in both years, suggest that the lower penetrance in the $P_1$, $F_1$ and $F_2$ populations grown for the first time in 1956 was genetically determined.

### (iii) *Discussion*

Many cases of incomplete penetrance involve variations in organ numbers, and can be explained by assuming variations in rates of organ formation, or in the time during which susceptible tissues remain in a reactive state (Goldschmidt, 1927). In this case, an existing petal divides at some early stage of its development. This may be envisaged as a continuation of the process of differentiation of the petal primordia, which in normal flowers stops when four petals have been laid down, but in abnormal flowers continues long enough for some of the petal primordia to divide again. This lengthening of the period during which petal primordia can differentiate could result from a hastening of differentiation in relation to other processes of

floral development, or from a lengthening of the period during which differentiation is possible. Either effect could be produced by variations in the amount of evocating substance which initiates differentiation. The correlations between the proportion of flowers and petals affected, and between penetrance and expressivity within families would then be expected.

The variation in the proportion of affected petals from time to time must be attributed to environmental variations, but whether these variations affect the floral embryos directly or through altering the internal environment of the plant is not known. They are apparently not an automatic consequence of the development of the plant, as no consistent pattern of occurrence could be detected. Nor is it surprising that the trait is genetically modified. The process of petal formation in affected plants must be delicately balanced or the abnormality would occur regularly. Variations in penetrance, with their consequent variations in dominance, could result from both genetic and environmental modification.

This character is apparently widespread in the *Cruciferae*, and the genetic situation underlying its appearance is similar in *Brassica napus* and *Raphanus sativa*.

It is not known, however, whether an homologous locus is involved in each species. The present situation might have arisen as a result of mutation followed by the assembling of a system of inhibitors which is occurring in these populations, or it might have arisen from the breakdown of such a system of inhibitors as a result of selection for some other characters. It may be significant that the anomaly has been seen only in cultivated members of the family.

Two main questions remain unanswered. They are the immediate cause of the appearance of the abnormality, which must be some substance produced in the plant or the developing flowers themselves, and the exact nature of the system of genetic modifiers. This material is not particularly suitable for either sort of enquiry. The absence of regularity in the appearance of affected flowers would render the first investigation difficult, while the labour involved in determining the phenotype of plants with a low proportion of abnormal flowers prohibits the use of the larger progenies necessary for a more comprehensive analysis.

## 5. SUMMARY

1. In populations of swede (*Brassica napus*) curly Kale (*B. oleracea*), and radish (*Raphanus sativa*) and evening scented stock (*Matthiola bicornis*) plants have been seen which bore, on some flowers, divided petals.

2. In the swede and radish the proportion of abnormal flowers varied from time to time on the one plant, though no consistent pattern of occurrence was detected.

3. The number of petals affected varied more on the radish than swede, and was correlated with the proportion of flowers affected.

4. In the swede and radish populations in which this abnormality was found, lines with varying proportions of abnormal plants, and of varying degrees of abnormality were isolated. The two characters were correlated. No true breeding normal plants were isolated in these populations, but on some plants the proportion of abnormal flowers was as low as $\frac{1}{2000}$.

5. It was concluded that variations in these populations are genetically influenced, some genotypes being more likely to produce abnormal flowers than others. At least two types of families, one producing a higher proportion of abnormal flowers than the other, were shown to occur in these populations.

6. In crosses between these abnormal swedes and plants of a population which is probably without this defect, results could be explained by assuming that one dominant gene with varying penetrance rates in the homozygous and heterozygous condition determined whether or not plants would, or could, produce abnormal flowers.

7. It was suggested that the gene, and its modifiers, act by increasing the production of the substance which initiates petal differentiation.

## 6. REFERENCE

GOLDSCHMIDT, R. B. 1927. *Physiologische Theorie der Vererbung.* Berlin : J. Springer. 247 pp.

# ENVIRONMENTAL EFFECTS ON THE DISTRIBUTION OF CHIASMATA AMONG NUCLEI AND BIVALENTS AND CORRELATION BETWEEN BIVALENTS

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## 1. INTRODUCTION

It is known from cytological evidence in a number of plants and animals that chiasma formation is not always independent in different bivalents, but may be correlated. Genetical evidence in *Drosophila* also indicates that crossing-over in different bivalents is correlated when inversions are present in heterozygous condition. A positive correlation between the chiasma frequencies of two bivalents means that when the number of chiasmata in one is low, it tends to be low in the other in the same nucleus ; high chiasma frequencies in one will be associated with high chiasma frequencies in the other. In the case of a negative correlation, a high number of chiasmata in one bivalent is associated with a low number in the other in the same nucleus.

When genetical mapping is done by selection of recombinants as in micro-organisms, correlations in crossing-over may have important effects on the observed frequencies of recombination. In a genetical study of such correlations, it is difficult to follow crossing-over in more than two bivalents simultaneously, owing to the limited number of marker genes which can be handled in a single experiment. Cytological studies of chiasma frequency possess the advantage that we can readily follow all bivalents.

In a sample of $n$ nuclei, each with $k$ bivalents (all of which are individually distinguishable), the variation in chiasma frequency over all bivalents can be partitioned as follows (Mather, 1936) :—

| Component of variation | Degrees of freedom |
|---|---|
| Between nuclei (internuclear) . . . . | $n-1$ |
| Between means for bivalents (interbivalent) . . | $k-1$ |
| Inherent . . . . . | $(k-1)(n-1)$ |

If the internuclear variance is equal to the inherent variance, there is no correlation between bivalents. If the internuclear variance is greater than the inherent variance, there is a positive correlation. If the internuclear variance is less than the inherent variance, there is a negative correlation (Mather, 1936). The internuclear variance measures the variation in the total numbers of chiasmata per nucleus in the sample, while the inherent variance measures the variation in the distribution of chiasmata among the bivalents of the individual nuclei.

This paper describes the effect of temperature on these variances and on the correlation between bivalents in two liliaceous plants, the bluebell, *Endymion non-scriptus*, and a diploid variety of hyacinth (Yellow Hammer).

## 2. MATERIAL

All the plants of *Endymion non-scriptus* used in this investigation came from Madingley Wood, near Cambridge. Group I bulbs were undergoing meiosis when collected on 21st December 1952. Group II bulbs were collected on 18th October 1952 and placed in pots of soil in controlled temperature chambers (see Elliott, 1955).

The hyacinth material and the temperature treatments given have been described previously (Elliott, 1955).

Chiasma frequencies were scored at first metaphase in temporary acetocarmine squashes of anthers.

## 3. METHOD OF ANALYSIS

In *Endymion non-scriptus*, the eight chromosomes of the haploid set are designated A to H in decreasing order of length (Darlington, 1926). At M−1 of meiosis, all the bivalents except B, C and D can be recognised individually (plate). B, C and D are treated as a single group. In the analysis of variance (*cf.* table, p. 429), the interbivalent variance therefore has only $k-3 = 5$ degrees of freedom. The internuclear variance still has $n-1$ degrees of freedom. If all remaining degrees of freedom contributed to the inherent variance, this would include an item with 2 degrees of freedom (out of the original $k-1$ of the interbivalent variance) for variation between the means of the three bivalents grouped together. However, we can separate an item with $2n$ degrees of freedom for all variation within nuclei between B, C and D. We thus replace the observed chiasma frequencies of B, C and D by three equal values in each nucleus. This leaves 5 $(n-1)$ degrees of freedom for the inherent variance, which measures the departures from the expected values of the observed values of the chiasma frequencies for A, E, F, G and H and the values, as obtained above, for B, C and D.*

| Component of variation | Degrees of freedom |
|---|---|
| Between nuclei (internuclear) | $n-1$ |
| Within nuclei | |
| Interbivalent (A, $\overline{\text{BCD}}$, E, F, G, H) | 5 |
| Between B, C and D | $\left\{\begin{matrix}2\\2(n-1)\end{matrix}\right\}$ $2n$ |
| Inherent | $5(n-1)$ |

At M−1 in diploid hyacinths, 4 L, 2 M and 2 S bivalents can be recognised, but the members of each group cannot be distinguished. For the analysis of whole nuclei, the variation within nuclei between the bivalents grouped together is again removed before finding the inherent variance, as follows :—

| Component of variation | Degrees of freedom |
|---|---|
| Between nuclei (internuclear) | $n-1$ |
| Within nuclei | |
| Between means for L, M and S | 3 |
| Between Ls | $3n$ |
| Between Ms | $n$ |
| Between Ss | $n$ |
| Inherent | $2(n-1)$ |

* I am indebted to Dr B. I. Hayman for this method of analysis.

## 4. RESULTS WITH *ENDYMION NON-SCRIPTUS*

Table 1 gives the mean chiasma frequency per nucleus and the internuclear and inherent variances for each plant, and table 2 tests of heterogeneity of the variances.

TABLE 1

*Analysis of variance of chiasma frequency and correlation between bivalents in* Endymion non-scriptus

| Plant no. | No. of cells ($n$) | Mean Xta per nucleus | Internuclear variance ($n-1$) d.f. | Inherent variance $5(n-1)$ d.f. | Correlation * |
|---|---|---|---|---|---|
| **Group I : Meiosis under natural conditions** | | | | | |
| MW 10 | 40 | 16·50 | 0·3397 | 0·3412 | o |
| MW 6 | 40 | 18·40 | 0·2532 | 0·3240 | o |
| MW 11 | 40 | 19·33 | 0·4513 | 0·3321 | o |
| MW 8 | 40 | 21·30 | 0·3667 | 0·2382 | + |
| MW 1 | 40 | 23·30 | 0·2770 | 0·3876 | o |
| **Group II : Meiosis at controlled temperatures** | | | | | |
| 1° 52/1/2 | 40 | 17·68 | 0·5083 | 0·3901 | o |
| 52/1/1 | 40 | 18·48 | 0·2756 | 0·3108 | o |
| 52/1/3 | 20 | 18·55 | 0·7299 | 0·2987 | + |
| 5° 52/5/2 | 40 | 16·38 | 0·3570 | 0·3298 | o |
| 52/5/3 | 40 | 16·93 | 0·3999 | 0·2753 | o |
| 52/5/1 | 40 | 17·50 | 0·6090 | 0·3393 | + |
| 15° 52/15/6 | 40 | 16·30 | 0·2385 | 0·4400 | − |
| 52/15/5 | 40 | 17·30 | 0·6615 | 0·3803 | + |
| 52/15/1 | 40 | 17·65 | 0·7038 | 0·3833 | + |
| 52/15/4 | 40 | 18·18 | 0·5634 | 0·3822 | o |
| 52/15/7 | 40 | 19·30 | 0·4372 | 0·4867 | o |
| 52/15/3 | 20 | 19·50 | 0·3618 | 0·3673 | o |
| 51/10/5 Anther 1 | 20 | 16·90 | 0·3803 | 0·2404 | o |
| 2 | 26 | 16·62 | 0·1408 | 0·2968 | − |
| 3 | 14 | 16·29 | 0·5275 | 0·1795 | + |

* + and − : correlations, positive or negative, significant at the 5 per cent. level.
o : no correlation between bivalents.

In four of the five plants which were undergoing meiosis under natural conditions (Group I), there is no correlation between the chiasma frequencies of the bivalents. In one plant there is a positive correlation (table 1). Both the internuclear and inherent variances for all five plants are homogeneous (table 2). As the ratio of the pooled variances indicates no correlation between bivalents, the plant with a positive correlation does not constitute a significant deviation from the population as a whole.

The experimental plants of Group II present a different picture.

Here a number of plants show a positive correlation between bivalents and others absence of correlation, and one plant shows a negative correlation between bivalents (table 1). Inspection of table 1 indicates that there is no relation between the type of correlation observed and the mean chiasma frequency of the plant.

The data for 51/10/5, a plant used in a temperature experiment in 1951, comprise scores for three separate anthers. Anthers 2 and 3 came from the same flower, anther 1 from a different flower on the same inflorescence. Anther 1 shows no correlation between bivalents; in anther 2 there is a negative correlation, and in anther 3 a positive correlation (table 1). The three anthers do not differ significantly in mean chiasma frequency.

TABLE 2

*Tests of heterogeneity of the variances in table 1*

| | Internuclear variances | | | Inherent variances | | |
|---|---|---|---|---|---|---|
| | $\chi^2$ | D.f. | P | $\chi^2$ | D.f. | P |
| Group I . . . | 4·09 | 4 | 0·5—0·3 | 8·94 | 4 | 0·1—0·05 |
| Group II | | | | | | |
| Within temperatures . | 24·35 | 9 | 0·01—0·001 | 12·09 | 9 | 0·3—0·2 |
| Between temperatures . | 0·60 | 2 | 0·8—0·7 | 13·90 | 2 | 0·01—0·001 |
| Anthers of plant 51/10/5 . | 8·71 | 2 | 0·001 | 5·00 | 2 | 0·1—0·05 |

The inherent variances are homogeneous for the plants within each temperature treatment in Group II and for the anthers of plant 51/10/5 (table 2), and are therefore not associated with the different types of correlation observed. The corresponding internuclear variances are, however, heterogeneous, and we observe that positive correlations are associated with large internuclear variances, and negative correlations with small internuclear variances. The striking feature of these results is that different types of correlation are associated with differences in internuclear variance without any change in inherent variance.

Two different effects of environment on the internuclear and inherent variances are evident in these results:

### (i) *An effect of temperature on the inherent variance*

The inherent variance measures the variation from cell to cell in the distribution of chiasmata among the bivalents of the nuclei. The variances for the plants at each temperature do not differ significantly, but there are differences between temperatures (table 2).

| Temperature | Mean inherent variance |
|---|---|
| 1° | 0·3332 |
| 5° | 0·3148 |
| 15° | 0·4066 |

There is an effect of temperature on the inherent variance, which is minimal at 5°, although the difference between the mean variances at 1° and 5° is not significant; the difference between them at 5° and 15° is significant.

The relation between the inherent variation and the variation of the individual bivalents in chiasma frequency is given by

$$\text{Inherent sum of squares} = \Sigma(a-\bar{a})^2+\tfrac{1}{3}\Sigma[(b+c+d)-(\bar{b}+\bar{c}+\bar{d})]^2 + \Sigma(e-\bar{e})^2+\ldots\ldots+\Sigma(h-\bar{h})^2-\text{Internuclear sum of squares}$$

where $a$, $b$, ... are the chiasma frequencies of the individual A, B, ... bivalents, and $\bar{a}$, $\bar{b}$, ... their means. We see from this equation that for a given amount of internuclear variation, an increase in the inherent variation is associated with increased variation in the chiasma frequencies of the individual bivalents. It may be that the observed effect of temperature on the inherent variance arises from an effect on the variability of the extent of chromosome pairing during meiotic prophase. At 15° pairing is less regular than at the lower temperatures; hence variation in the chiasma frequency of the individual bivalents is greater, and there is a corresponding increase in inherent variance.

#### (ii) *An effect of some unknown factor on the internuclear variance*

The internuclear variance measures the range in numbers of chiasmata per nucleus of the pollen mother cells. The variances are heterogeneous between plants in each temperature treatment, but not between temperatures (table 2). The heterogeneity of the internuclear variances for the three anthers of plant 51/10/5 clearly cannot be due to genetical causes, and it may well be that the heterogeneity between plants at each temperature in Group II is likewise not due to genotypic differences, since the variances are homogeneous in Group I. Something has happened to the experimental plants which has upset the normal control of the amount of variation between nuclei in many of them, irrespective of the temperature at which they were placed. Different parts of one plant may even be differently affected. We postulate that these changes in the internuclear variance are the result of environmental effects on an extranuclear factor acting on the nuclei of the pollen mother cells and determining the distribution of chiasmata among them.

### 5. RESULTS WITH *HYACINTHUS*

#### (i) *Analysis of whole nuclei*

Table 3 gives the numbers of bulbs, flowers and nuclei scored at each of the temperatures used. The analysis of variance was carried out separately for each flower, and table 3 gives the mean internuclear and inherent variances (weighted according to the numbers of degrees of freedom for each sample). Table 4 gives tests of heterogeneity of the variances for the flowers at each temperature and between temperatures.

The main interest of the results with hyacinths is in the pooled results for the different temperatures. The internuclear variances do not differ significantly between the three temperatures 20°, 15° and

TABLE 3

*Summary of analysis of variance of chiasma frequency and correlation between bivalents in* Hyacinthus

| Temperature | Numbers of | | | Internuclear variance | Inherent variance | Correlation |
|---|---|---|---|---|---|---|
| | Bulbs | Flowers | Nuclei | | | |
| 20° | 4 | 8 | 375 | 0·4068 | 0·3600 | 0[a] |
| 15° | 4 | 8 | 400 | 0·4720 | 0·3227 | +[b] |
| 10° | 4 | 8 | 371 | 0·4627 | 0·4339 | 0[c] |
| 5° | 6 | 15 | 600 | 1·9642 | 0·5133 | + |

[a] Positive correlation in one flower, and negative correlation in one.
[b] Positive correlations in four flowers.
[c] Negative correlation in one flower.

10°; at 5° the variance is much greater. The inherent variances for the four temperatures are, however, heterogeneous. As in the bluebell, there is an effect of temperature on the inherent variance, which is here minimal at 15°. The differences between the mean

TABLE 4

*Tests of heterogeneity of the variances in flowers of* Hyacinthus

| | Internuclear variances | | | Inherent variances | | |
|---|---|---|---|---|---|---|
| | $\chi^2$ | D.f. | P | $\chi^2$ | D.f. | P |
| Between temperatures | | | | | | |
| 20°, 15°, 10°, 5° . | 452 | 3 | <0·001 | 70·14 | 3 | <0·001 |
| 20°, 15°, 10° . . | 2·43 | 2 | 0·3—0·2 | | | |
| Within temperatures | | | | | | |
| 20° . . . | 18·65 | 7 | 0·01—0·001 | 7·10 | 7 | 0·5—0·3 |
| 15° . . . | 5·52 | 7 | 0·7—0·5 | 7·09 | 7 | 0·5—0·3 |
| 10° . . . | 12·14 | 7 | 0·1—0·05 | 16·31 | 7 | 0·05—0·02 |
| 5° . . . . | 36·97 | 14 | <0·001 | 19·19 | 14 | 0·2—0·1 |

variances at 5° and 10° and at 10° and 15° are significant, but that between them at 15° and 20° is not significant.

At each of the temperatures 20° and 10° the mean internuclear and inherent variances do not differ significantly, so there is no correlation between bivalents for these temperatures as a whole. At 15° the internuclear and inherent variances differ, indicating a positive correlation (table 3). It is to be noted that while the internuclear variances are the same for the three higher temperatures, the inherent variance is lower at 15° than at 20° and 10°. We may therefore

associate the positive correlation at 15° with a change in the inherent variance, in contrast to the results at one given temperature in bluebells.

At 5°, the analysis of variance indicates a positive correlation between the chiasma frequencies of the bivalents, and this bears out the observation of a positive correlation in their pairing behaviour. For each of the chromosome types, L, M and S, more nuclei have all univalents or all bivalents than is expected by chance (Elliott, 1955).

Within temperatures, the inherent variances are homogeneous, as in bluebells, although they are heterogeneous at 10°.

Again within temperatures, the internuclear variances are heterogeneous at 20° and 5°, and homogeneous at 15° and 10°. That is to say, at 20° and 5° there is more variation in chiasma frequency between the nuclei in some samples of pollen mother cells than in others, while at 15° and 10° the variation between the nuclei of the p.m.c. does not differ significantly between samples. Thus while temperature does not affect the mean variance of the distribution of chiasmata between nuclei, except at 5°, we may say that it strongly affects the " stability " of this distribution.

At 20° the negative correlation observed in one flower is associated with a low internuclear variance, and the positive correlation in another with a high internuclear variance. The negative correlation in one flower at 10° is also associated with a low internuclear variance. Thus within temperatures the relation between different types of correlation is the same as that observed in the bluebells.

### (ii) *Interrelationships of the grouped bivalents*

The inherent variance as calculated above measures the variation from cell to cell between the three classes of bivalents, L, M and S. We now consider the relationships between the bivalents in each of the three groups. The variation between and within nuclei for each group has been calculated in the usual way for four of the flowers at each temperature, and table 5 gives the pooled variances, and the type of correlation between the bivalents of each group for the temperature as a whole.

The variation between bivalents within nuclei differs significantly between temperatures (table 5), but unlike the inherent variance discussed above, it is less at 20° than at 15° for each class of bivalent, significantly so for the L and M chromosomes. It cannot be said whether this represents a real difference between the variance of the distribution of chiasmata to the three groups and that to the bivalents of the groups. Clearly, however, the variation between bivalents is affected by temperature.

The variation between nuclei for the L and M bivalents does not differ significantly between the three higher temperatures (table 5), as is the case also for the whole nuclei, but for the S bivalents there is more variation between nuclei at 10° than at 15° and 20°. This may be explained by the frequent failure of the S chromosomes to form

bivalents at low temperature, which is experienced by the L and M chromosomes only at 5°.

## 6. THE ROLE OF DIFFERENT BIVALENTS IN CORRELATED SYSTEMS IN *ENDYMION NON-SCRIPTUS*

From a genetical standpoint, the correlations between the bivalents of the nucleus as a whole are of less interest than correlations between the chiasma frequencies of one particular bivalent and another or

TABLE 5

*Summary of analysis of variance of chiasma frequency of and correlation between the L, M and S bivalents of* Hyacinthus

| Temperature | L | | | M | | | S | | |
|---|---|---|---|---|---|---|---|---|---|
| | Between nuclei | Within nuclei | Correlation * | Between nuclei | Within nuclei | Correlation * | Between nuclei | Within nuclei | Correlation * |
| 20° | 0·5889 | 0·5016 | 0 | 0·3603 | 0·2450 | +[a] | 0·1074 | 0·1375 | − |
| 15° | 0·6417 | 0·6217 | ◻ | 0·3581 | 0·3625 | ◻ | 0·1328 | 0·1500 | ◻ |
| 10° | 0·7445 | 0·8954 | −[b] | 0·3740 | 0·4100 | ◻ | 0·2544 | 0·2800 | 0[b] |
| 5° | 2·0703 | 0·7625 | + | 0·7505 | 0·4025 | + | 0·6351 | 0·2900 | + |
| Heterogeneity between temperatures | | | | | | | | | |
| 20°, 15°, 10°, 5° $\chi^2$ [3] | ... | 55·9* | ... | ... | 16·1* | ... | ... | 45·8* | ... |
| 20°, 15°, 10° $\chi^2$ [2] | 2·78 | ... | ... | 0·11 | ... | ... | 41·4* | ... | ... |

[a] Significant positive correlation in two of the four flowers.
[b] Significant negative correlation in one of the four flowers.
* Significant values at 5 per cent. level.

group of other bivalents. *Endymion non-scriptus* is an exceptionally favourable organism for studies of chiasma frequency as we can recognise five of the eight chromosomes at M−1 (plate). The following analyses have been made to determine the behaviour of the individual types of bivalents in a correlated system.

Correlation coefficients have been calculated (i) for the chiasma frequencies of the bivalents A, E, F, G and H considered in pairs, (ii) for the numbers of chiasmata in a particular type of bivalent (including B, C and D as a group) compared with the totals in the remaining bivalents (table 6). This has been done in plant 52/15/6 and anther 2 of plant 51/10/5, in both of which the previous analysis of variance indicated a negative correlation between bivalents for the nucleus as a whole, and in 52/15/5 and anther 3 of 51/10/5, where there is a positive correlation. The use of correlation coefficients is open to objection, since some bivalents have only two different

numbers of chiasmata in a sample of nuclei. In a number of cases therefore the independence of the bivalents has been examined by the exact method (Freeman and Halton, 1951). With these relatively small numbers of nuclei, few of the correlations are expected to be

TABLE 6

*Correlations observed (whether positive or negative) between the chiasma frequencies of the bivalents of* Endymion non-scriptus *considered in pairs, and between the numbers of chiasmata in one type of bivalent and the total in the remaining bivalents. Significant correlations are indicated by an asterisk*

| | 52/15/6 | | | | | | 52/15/5 | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | A | BCD | E | F | G | H | A | BCD | E | F | G | H |
| A | ... | ... | − | − | − | + | ... | ... | − | + | + | + |
| E | ... | ... | ... | + | − | − | ... | ... | ... | + | − | − |
| F | ... | ... | ... | ... | − | − | ... | ... | ... | ... | + | − |
| G | ... | ... | ... | ... | ... | − | ... | ... | ... | ... | ... | + |
| Remainder of nucleus | −* | −* | − | −* | − | − | +* | + | + | + | + | − |
| | 51/10/5, anther 2 | | | | | | 51/10/5, anther 3 | | | | | |
| | A | BCD | E | F | G | H | A | BCD | E | F | G | H |
| A | ... | ... | + | − | + | −* | ... | ... | + | + | + | + |
| E | ... | ... | ... | + | + | − | ... | ... | ... | o | − | + |
| F | ... | ... | ... | ... | + | + | ... | ... | ... | ... | + | − |
| G | ... | ... | ... | ... | ... | − | ... | ... | ... | ... | ... | + |
| Remainder of nucleus | − | −* | + | − | − | −* | + | + | + | +* | +* | − |

significant. In table 6, the symbols + and − indicate whether the correlation is positive or negative, and an asterisk that the value of the correlation coefficient is significant at the 5 per cent. level.

For the bivalents considered in pairs, the correlation is in the same direction as that for the nucleus as a whole in 25 cases, and of opposite sign in 14. One of the 40 correlation coefficients is significant. For the comparison of one type of bivalent with the remaining bivalents, the correlation is in the same direction as that for the nucleus as a whole in 21 of the 26 cases, and 8 of the correlation coefficients are significant. Thus while the correlation between individual bivalents is generally not significant, the effects are predominantly in one direction and tend to become significant when more bivalents are considered together. In contrast to the above, in two samples where the analysis of variance indicated no correlation between bivalents, none of the correlations of table 6 is significant.

Now the variances of the chiasma frequencies of the different bivalents are heterogeneous (table 7 ; $\chi^2_{(5)} = 39{\cdot}73$, $P < 0{\cdot}001$). (No account of this has been taken in the analysis of variance of table 1.) Bivalent E has the lowest variance. Table 7 also gives the mean chiasma frequency and the variance of the distribution for the separate arms of the bivalents. It will be noticed in table 7 that there is a connexion between the variance of the distribution of numbers of

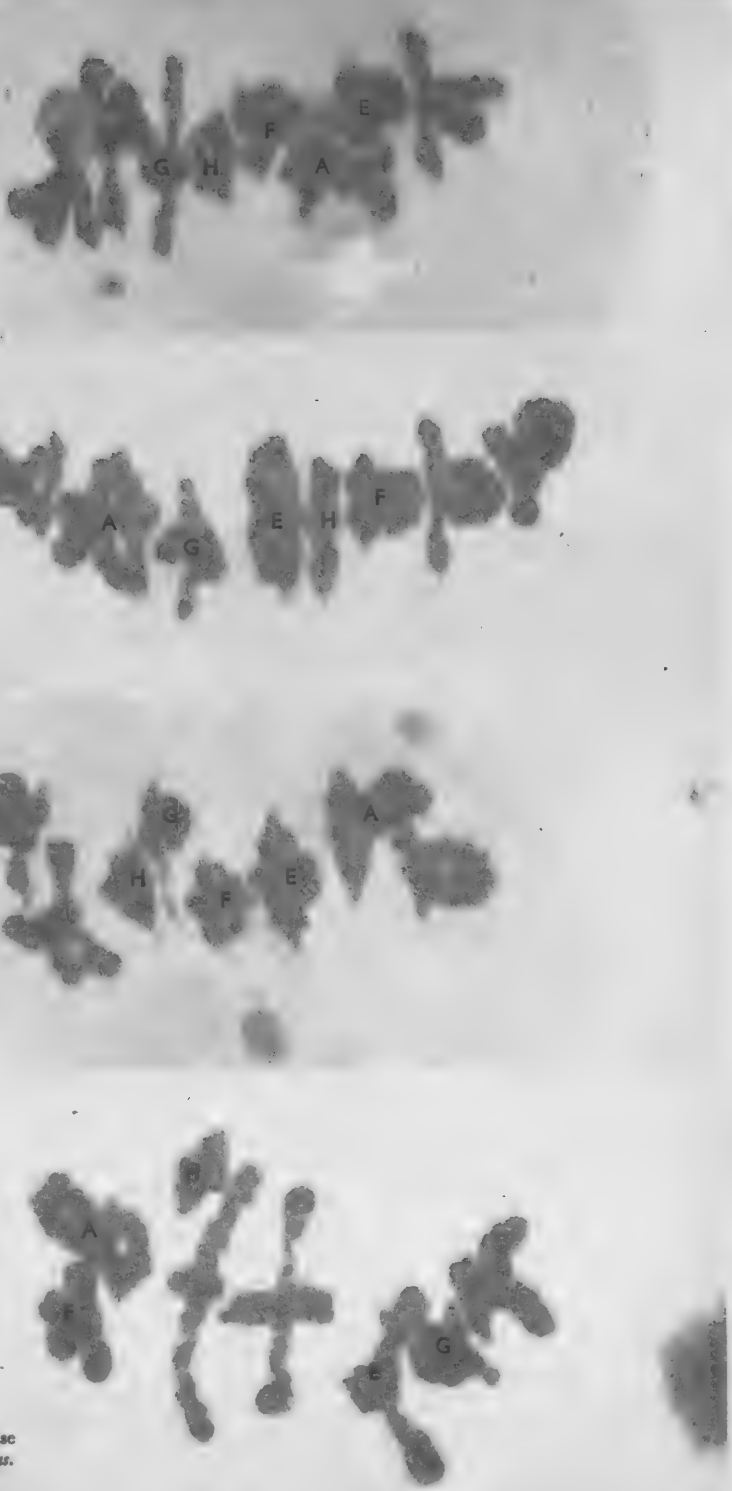
TABLE 7

*Mean chiasma frequencies and variances of the distributions for the whole bivalents and for their separate arms in* Endymion non-scriptus, *plant MW 6, together with mitotic lengths of the chromosomes as a percentage of the length of the haploid set*

| Chromosome (arm) | Length | Mean Xa frequency | Variance |
|---|---|---|---|
| H | 7·3 | 1·65 | 0·2333 |
| G | 9·0 | 1·68 | 0·2763 |
| F | 10·8 | 2·15 | 0·2846 |
| E | 13·1 | 2·13 | 0·1122 |
| BCD | 13·9 | 2·64 | 0·5512 |
| A | 18·0 | 2·88 | 0·4711 |
| G short | 0·9 | ■ | 0 |
| BCD short | 2·0 | 0·37 | 0·2342 |
| H short | 2·4 | 0·65 | 0·2333 |
| F short | 3·2 | 0·93 | 0·0712 |
| H long | 4·9 | 1·00 | ■ |
| A short | 5·2 | 0·93 | 0·0712 |
| E short | 6·0 | 1·00 | 0 |
| E long | 7·1 | 1·13 | 0·1122 |
| F long | 7·6 | 1·23 | 0·1788 |
| G long | 8·1 | 1·68 | 0·2763 |
| BCD long | 11·9 | 2·28 | 0·4532 |
| A long | 12·8 | 1·95 | 0·3051 |

chiasmata per arm and their mean. Arms with a mean of one form one chiasma with great regularity. The variance of the whole bivalent E is the lowest of the six because E is the chromosome with most nearly equal arms and the chiasma frequency of each arm is about one. In the longer arms which may have more than one chiasma, the variance increases with the mean.

Considering the correlations between individual bivalents in table 6, bivalent A showed a correlation with the other individual bivalents of the same sign as that for the nucleus as a whole in 12 out of 16 cases, G and H each in 11 out of 16 cases, F in 9 out of 15 and E in 7 out of 15 cases. Thus, while none of the ratios 12 : 4 etc., deviates significantly from 1 : 1, there is a suggestion that the occurrence of correlations between the bivalents of a nucleus is more dependent on the behaviour of the long and short chromosomes than on those of intermediate length. This is clearly related to the observation that the long arms of A and G and the short arm of H have a greater variance of chiasma frequency than the arms of E and F (table 7).




*Plate*

First meiotic metaphase in *Endymion non-scriptus*. ×1500.

## 7. SUMMARY AND CONCLUSIONS

The variances of the distributions of chiasmata among nuclei and among the bivalents of the nuclei are differently affected by environment, suggesting they are independent and determined by different physiological processes. This is in line with the observation (Rees and Thompson, 1956) that in rye they are at least in part independently determined genetically.

Different types of correlation between the chiasma frequencies of the bivalents may be observed when one of the variances is altered without any change in the other. The results with hyacinths are especially important ; here, changes with temperature in the inherent variance (between bivalents), unaccompanied by changes in the internuclear variance, were associated with different types of correlation. In *Endymion non-scriptus*, bulbs at the same temperature, and even different anthers in the same bulb, showed differences in internuclear variance, which, unaccompanied by changes in the inherent variance, were associated with different types of correlation.

Finally, as illustrated in *Endymion*, no correlation is expected between different bivalents if the variance of the chiasma frequency of one or both of them is low. If the variance for both bivalents is high, a significant correlation may occur.

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# CYTOLOGICAL ABNORMALITIES IN *PENNISETUM DUBIUM*

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## 1. INTRODUCTION AND MATERIALS

THE genus *Pennisetum* consists, cytologically, of two groups of species, one with a basic chromosome number of $x = 7$ and the other with $x = 9$ (Darlington and Wylie, 1956). The annual large-seeded *P. typhoides* ($2n = 14$) has been used in hybridisation programmes in an endeavour to breed a large-seeded perennial for use in ley farming (Gildenhuys, 1950). It was crossed with perennial *P. purpureum* ($2n = 28$), the only other known species in the $x = 7$ group.

In a search for other perennial species to be used for crossing, the senior author collected a type in the Ermelo district of the Eastern Transvaal. He found the following chromosome numbers in plants of this collection ; $2n = 56, 63, 66, 67, 70$ and 86 (Gildenhuys, 1951).* These plants were remarkably uniform both in their natural habitat and in progenies raised from seed. On crossing one plant of his collection onto *P. typhoides* he obtained one hybrid plant which had only 21 chromosomes. It had received 14 chromosomes from a parent with at least 56. The behaviour of the polyploid parent became a matter of interest and it is this behaviour, reported below, which has led us to suggest for this type the *nomen P. dubium.*†

## 2. OBSERVATIONS

### (i) *Mitosis*

Nine clones grown in pots in the field and numerous natural seedlings germinated in an incubator at 25° C. all showed varying numbers of chromosomes in the same root tip. Complements varying in number from 14 to 84 (161 cells) were observed, the most frequent number being 66 (fig. 1A). Cell size increased with chromosome number.

To obviate the possibility of over-squashing (Walker *et al.*, 1954), counts from intact cells only were recorded.

* This type was identified by Professor A. P. Goossens as *P. sphacelatum*. Since then, however, Dr Hubbard and others have classified it as *P. macrourum*. Crook (1955) pointed out the difficulties of distinguishing between these two species, but according to Mr D. Killick specimens at Kew, where this grass was sent for matching, are distinct. The chromosome number of *P. sphacelatum* has not yet been determined. The number given by Darlington and Wylie for *P. macrourum* is $2n = 45$, as determined by Avdulov. Mr D. Killick, who checked on Avdulov's original publication, states that this number should read $2n = 54$ and also points out that there cannot be certainty that Avdulov's material was correctly named.

† Root tips were stained and squashed in aceto-carmine with ferric chloride, anthers in acetic-orcein. Both were fixed in Carnoy's (6 : 3 : 1). Root tips were sometimes prefixed for 2½ hours in a saturated aqueous solution of α-bromonaphthalene.

Cells with several nuclei were also observed. The timing of nuclear divisions in these cells was often irregular. In the same cell

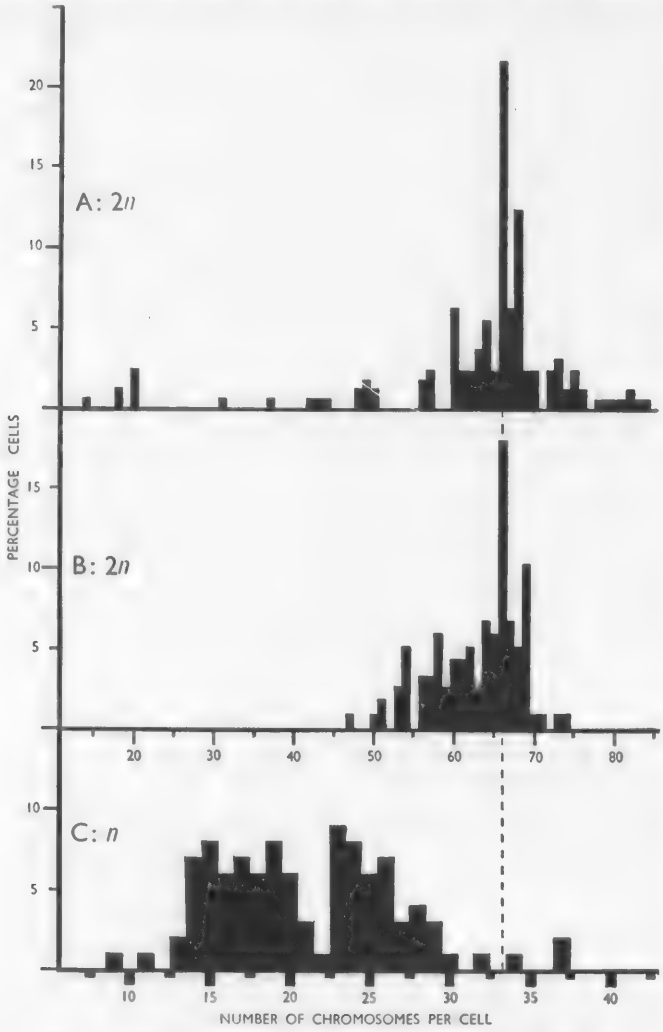


FIG. 1.—Frequency distributions of different chromosome complements per cell in root tips and anthers of *P. dubium*.

A. Root tip squashes (2*n*).
B. P.M.C. squashes—Meiosis I (2*n*).
C. P.M.C. squashes—Meiosis II (*n*).

one or more nuclei might be in the resting stage, whilst the remainder were in any one of the different phases of division (fig. 2A-B). The

different nuclei in a cell contained chromosomes ranging in number from a few (14) to as many as 66.

Again, there might be in the same cell groups of chromosomes at all phases of mitosis. Such groups could be distinguished from those formed by the simultaneous division of more than one nucleus in the same cell. This happened both in pretreated and in untreated material. The number of groups was mostly 2 to 4 (fig. 2D), but in rare cases 5 or 6. In some cells single chromosomes or very small groups of chromosomes appeared to be scattered at random in the

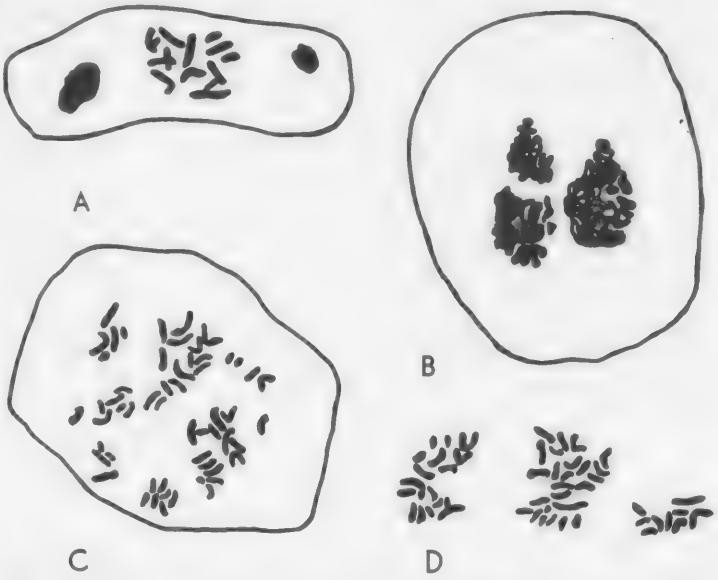


FIG. 2.—Mitotic abnormalities in *Pennisetum dubium*. A-B. Timing of division in R.T. cells with more than one nucleus. A. Two resting nuclei and one metaphase nucleus with 14 chromosomes. B. Three late prophase nuclei of unequal size. C-D. Group formation of chromosomes. C. Many small groups and scattered individuals at metaphase. D. Three groups at metaphase. All figs. ×1260.

cytoplasm, away from the main groups (fig. 2C). In cells with a large number of groups it was occasionally possible to count the number of chromosomes and they varied from 2 to 22 per group in equal or unequal numbers.

Equatorial views of metaphase occasionally showed groups not orientated on the equator. Tripolar, quadripolar and split spindles could be seen at anaphase, and lagging chromosomes were also observed. Cells with telophase nuclei often also had micronuclei and/or individual chromosomes scattered in their cytoplasm. Cell walls were rarely formed.

Very few cell divisions were found in the root tips of two clones and in these multinucleate cells appeared to be more prevalent than in the remaining clones. Table 1 shows the frequency of all types of abnormalities at all phases of mitosis in 5 different clones.

TABLE 1

*Frequencies of abnormal mitotic cell divisions in* P. dubium *: Numbers of cells examined in each phase are shown in brackets*

| Plant no. | Percentage of abnormal cells | | | Total cells examined | Means |
|---|---|---|---|---|---|
| | Prophase | Metaphase | Anaphase and Telophase | | |
| 5 | (49) 51·0 | (91) 25·3 | (69) 20·3 | *209* | 29·7 |
| 3 | (11) 54·5 | (24) 20·8 | (20) 40·0 | *55* | 34·5 |
| 4 | (115) 53·1 | (184) 23·9 | (115) 45·2 | *414* | 37·9 |
| 11 | (37) 62·2 | (59) 28·8 | (49) 44·9 | *145* | 42·8 |
| 6 | (24) 58·3 | (60) 40·0 | (65) 49·2 | *149* | 47·0 |
| Means | (236) 54·7 | (418) 27·1 | (318) 40·3 | *972* | 38·1 |

### (ii) *P.M.C. meiosis*

Abnormalities similar to those in the root tips were also encountered at meiosis, but they were less intense and less frequent (table 2).

TABLE 2

*Frequencies of abnormal meiotic cell divisions in* P. dubium *: Numbers of cells examined in each phase are shown in brackets*

| Plant no. | Percentage of abnormal cells | | | | | | Total cells examined | Means |
|---|---|---|---|---|---|---|---|---|
| | Metaphase | | Anaphase | | Telophase | | | |
| | I | II | I | II | I | II | | |
| 3 | (200) 3·5 | (200) 0·0 | (67) 0·0 | (91) 1·1 | (200) 0·0 | (200) 0·0 | *958* | 0·84 |
| 5 | (217) 6·9 | (200) 1·5 | (69) 4·4 | (86) 3·5 | (200) 0·0 | (200) 0·0 | *972* | 2·47 |
| 11 | (202) 2·5 | (209) 4·3 | (33) 6·0 | (68) 4·4 | (200) 1·0 | (178) 1·6 | *890* | 2·70 |
| 6 | (500) 11·2 | (507) 1·4 | (104) 3·8 | (127) 5·5 | (161) 0·6 | (301) 0·3 | *1700* | 4·47 |
| Means | (1119) 7·42 | (1116) 1·70 | (273) 3·30 | (372) 3·76 | (761) 0·39 | (879) 0·46 | *4520* | 2·92 |

Two or even three nuclei occurred in certain P.M.C's. These cells obviously arose from abnormal pre-meiotic mitoses which resulted in two types :—(i) binucleate cells which arose through failure of cell-division and (ii) those in which one of the nuclei of binucleate cells had undergone a reduction division, thus producing two reduced interphase nuclei and one persistent pre-meiotic resting nucleus.

These two types could be distinguished by the sizes and positions of their nuclei. The timing of the nuclear divisions in such cells was almost invariably irregular (fig. 3D), and none of them produced countable chromosome plates. The few nearly countable ones revealed upwards of 50 in the same cell with one or two resting nuclei. Interphase dyads had no more than two nuclei per cell. Some of these were simultaneously dividing with two metaphase plates, one above the other in the same cell. Rarely one cell of a dyad contained a normal nucleus whilst the other contained only a micronucleus surrounded by stained material.

Tripolar and quadripolar spindles also occurred at anaphase of both divisions. Such spindles would account for the presence of two nuclei in cells of interphase dyads. From the sizes of these nuclei they could not be confused with micronuclei following lagging.

Groups of chromosomes were also observed at all phases of both divisions. Here, however, two complications arose. First, the occurrence of sticky chromosomes precluded the possibility of distinguishing clearly, in first divisions, between the types of groups found at mitosis and the type formed as a result of stickiness or a combination of both (fig. 3C). Second, lack of association and also of co-orientation of both bivalents and univalents together with stickiness could equally well account for the presence, in certain cells, of individual chromosomes and small groups scattered away from the main groups. Stickiness was more pronounced in second divisions, but here, groups similar to those at mitosis could, in certain cases, be distinguished, uncomplicated by chiasma associations.

Stickiness frequently led to clumping (fig. 3C). In first division preparations, clumps often occurred in the same cell as distinguishable univalents, bivalents, etc. Stickiness persisted through interphase in which many preparations had the two nuclei joined by chromatin material in the form of either a broad band (fig. 3A) or thin extended threads curling in different directions through the cytoplasm (fig. 3B). Chromatin material joining sticky interphase nuclei was distinct from inversion bridges.

*Chromosome numbers.* As a result of stickiness and also of variation in chromosome numbers, it was often difficult to determine the associations and hence the chromosome numbers in first divisions. Consequently, the chromosome numbers in first P.M.C. divisions were determined entirely from normal or nearly normal early anaphases. These numbers varied from 47 to 74 in 117 cells, the most frequent number again being 66 (fig. 1B). In dyad cells, in which stickiness was less severe, the numbers varied from 9 to 37 (101 cells), the most frequent number being 23 (fig. 1C).

*Chromosome associations and distribution.* From P.M.C.'s undergoing normal or near normal first divisions the chromosome associations in diakinesis and MI reflected in table 3 were obtained.

The lowest degree of association was in a cell with 27 I + 19 II = 65. Four cells (7·8 per cent.) showed no univalents. In these the associations were :—

2 III + 30 II = 66
1 III + 31 II = 65
33 II = 66 and
1 VI + 1 IV + 26 II = 62 (the only cell containing a sexivalent).

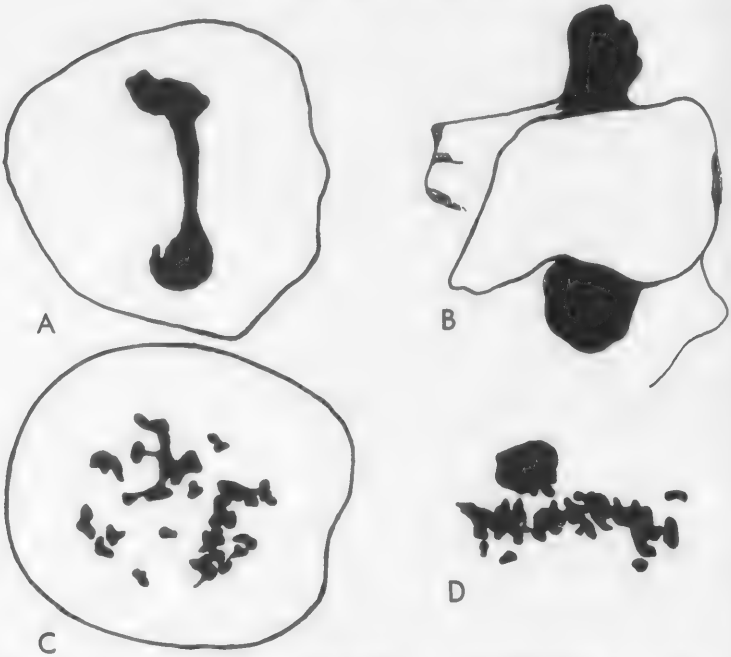


FIG. 3.—Meiotic abnormalities in *Pennisetum dubium*. A-C. Stickiness at meiosis I in P.M.C.'s. A-B. Sticky interphase nuclei joined by chromatin material in broad bands (A) or in thin curling extended threads (B). C. Chromosomes joined and aggregated into groups at MI. D. Timing of division in P.M.C. with more than one nucleus—one resting and one MI nucleus. All figs. × 1260.

Eighteen cells (35·3 per cent.) contained less than 5 univalents.

Univalents divided either at first or at second division. Bridges occurred in 70·5 per cent. of first divisions (table 4). They rarely appeared at second division.

In certain MI plates there was a lack of co-orientation of bivalents, which lay scattered in the cytoplasm whilst others were co-orientated. Occasionally however some were found to divide quite normally after the others had nearly reached the poles. Those which failed to orientate by late AI then migrated to the poles as undivided bivalents.

At late TI they were sometimes still undivided and lying outside the nuclei. They were not seen to persist as bivalents into second

TABLE 3

*Chromosome associations in P.M.C.'s at diakinesis and MI in* P. dubium

| Associations | VI | IV | III | II | I | 2*n* | Total cells examined |
|---|---|---|---|---|---|---|---|
| Totals . . | 1 | 21 | 12 | 1441 | 309 | 3317 | 51 |
| Means . . | *0·02* | *0·41* | *0·24* | *28·25* | *6·06* | *65·04* | |
| Range . . | 0-1 | 0-2 | 0-3 | 17-33 | 0-27 | 50-73 | |

division. In AII, however, there were occasionally undivided univalents, and they may have been the products of bivalents which had divided in the second division.

TABLE 4

*Frequencies of bridges at first meiotic divisions of* P. dubium

| Number of bridges . | 0 | 1 | 2 | 3 | 4 | 5 | Mean = 1·44 |
|---|---|---|---|---|---|---|---|
| Number of cells . . | 31 | 26 | 28 | 12 | 7 | 1 | Total = 105 |

Chromosome distribution at anaphase varied greatly. In cells with no laggards this variation was of the order of a 21 : 35 distribution to an even distribution as high as 33 : 33 for AI and 24 : 39 to even

TABLE 5

*Frequencies of laggards or micronuclei in meiosis of* P. dubium

| Phase | Number of laggards or micronuclei per cell | | | | | | | | | | | | | | | | | | | No. of cells | Means |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | | |
| Inter | 57 | 101 | 112 | 88 | 60 | 33 | 33 | 7 | 9 | 11 | 4 | 5 | 7 | 1 | 5 | 1 | 1 | 0 | 3 | *538* | 3·27 |
| TII | 103 | 132 | 102 | 69 | 55 | 18 | 30 | 10 | 9 | 3 | 4 | | | | | | | | | *535* | 2·32 |

(24 : 24 to 33 : 33) for AII. At interphase 89·4 per cent. and at TII 80·7 per cent. of cells showed laggards or micronuclei and these varied in number from 0 to 18 in the former and 0 to 10 in the latter phase (table 5).

Cell walls were usually found at late TI or later. Immediately cytokinesis had set in cells separated easily with very slight pressure.

*Tetrads and pollen.* Tetrads almost invariably had one or more micronuclei scattered in their cytoplasm. Other than size differences, the only abnormality which occurred at this stage was the presence of an occasional triad or a triad with a shrunken fourth cell containing a micronucleus surrounded by darkish material.

Pollen collected immediately after dehiscence revealed no significant differences between plants in either the percentage of stainable pollen or the sizes of the pollen grains. Of 4603 grains 28·0 per cent. did not stain as compared with 13·3 per cent. (1290 counted) in the diploid *P. typhoides*. The grains varied in size from 19 to 33 divisions on the ocular micrometer disc (mean = 25·9) as against 20 to 25 (mean = 22·6) for *P. typhoides* (table 6).

TABLE 6

*Frequencies of pollen grains of different sizes in* P. typhoides *and* P. dubium

| Species | Sizes in divisions of ocular micrometer | | | | | | | | | | | | | | | Means |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31 | 32 | 33 | |
| *P. typhoides* . | | 1 | 7 | 23 | 11 | 6 | 5 | | | | | | | | | 22·55 |
| *P. dubium* . | 2 | 13 | 15 | 26 | 45 | 59 | 125 | 85 | 82 | 58 | 45 | 50 | 9 | 3 | 2 | 25·94 |

## 3. ABNORMALITY AT MITOSIS

The mitotic abnormalities appear to be of three main kinds :

(1) *Delayed cytokinesis in otherwise normal cell divisions.* If the chromosome number of this grass is taken as $2n = 66$ then the highest number observed ($2n = 84$) indicates that cell-wall formation seldom fails altogether. The presence of a large nucleus in the same cell as about 66 metaphase chromosomes was the nearest approach to a cell with a doubled number that was observed. Multinucleate cells could, of course, have arisen through failure of cytokinesis, but they may also be the result of the other abnormalities discussed below. It seems more likely that cytokinesis seldom fails altogether but that it is delayed until subsequent division of the chromosomes. Because of the high frequency of abnormal anaphases, telophases and prophases as compared with metaphases (table 1), it is thought that it generally sets in after the first subsequent mitotic prophase. Table 1 includes as " abnormal " also normal divisions of more than one nucleus per cell.

(2) *Spindle abnormalities.* Although the presence of a spindle was not demonstrated, the discontinuous arrangement of chromosomes on equatorial plates (fig. 2c) could be the result of a discontinuous or incompact spindle as described by Darlington and Thomas (1937)

in meiosis of a *Festuca-Lolium* derivative. Tripolar and split or quadripolar spindles are evidently present in some cells. In cases where three nuclei appeared in the same somatic cell, one was often very much larger than the other two (fig. 2B), indicating the possibility that a tripolar spindle and failure of cell-wall formation after telophase may have been the cause. Owing to the fact that cytokinesis was so rare, an actual reduction in chromosome number due to spindle abnormalities was never observed. That this type of abnormality may lead to variation in chromosome number is, however, evident.

(3) *Group formation of chromosomes.* Over-squashing (Walker *et al.* l.c.) could well be the reason for the presence of groups, especially when these groups are separated by cytoplasmic " breaks ". In this material, however, numerous apparently intact cells without such breaks were observed to contain groups of chromosomes. The possibility that the chromosomes could be irregularly distributed after cytokinesis in such cells is apparent.

These abnormalities are similar to those described by Snoad (1955) in *Hymenocallis calathinum.* Whether they are all due to a single basic cause which upsets the spindle (Vaarama, 1949 and Snoad, l.c.) or whether they arise from separate causes (Östergren, 1950) cannot be stated without inducing them experimentally, as has been done by Huskins *et al.* (1950) and his school when they propounded their ideas on " somatic meiosis ", " reductional grouping " and " segregational reduction". Nevertheless, noteworthy facts are that they exhibit a wide range and that they occur naturally with a high frequency.

The views of Wilson *et al.* (1952) regarding the " semi-separable zones of activity " of the spindle might well explain different manifestations of an abnormality which could be induced extraneously, but which could, as appears in this case, also arise from the genotype. In fact, as Sachs (1954) has pointed out, not only can subdiploid variation be caused by environmentally controlled mitotic abnormalities, but certain gene combinations can cause distinct abnormalities such as split spindles in both mitosis and meiosis, multipolar spindles at meiosis and so on. In this case environmental agencies could be excluded, as the same abnormalities occurred in root tips of seedlings germinated under normal conditions.

## 4. ABNORMALITY AT MEIOSIS

Abnormalities similar to the three types described above also arise at meiosis. Added to these there are the stickiness and the abnormal reduction after the first meiotic divisions. Stickiness has been observed in many organisms by different workers. Evidence of the causes of this abnormality is scarce and vague. Beadle (1932) attributed stickiness in *Zea mays* to a single recessive gene. Walters (1950) stated, without further reference, that " . . . interspecific grass hybrids frequently have sticky meiotic chromosomes and this condition may

also be influenced by environmental conditions". Ono (1951) explained similar phenomena in intergeneric hybrids in Cichorieæ by his "hypothesis of residual affinity", whereby it is assumed that in nuclei of heterogeneous origin the presence of homologous sections, large or small, in different chromosomes, will cause secondary association to the extent of forming netlike configurations at early diakinesis. Recently Davies (1956) pointed out that stickiness often occurs in both meiotic divisions in species of plants and insects which lack localised centromeres. In almost all reported cases stickiness is accompanied by breakage or loss of chromosomes. The occurrence of stickiness at both meiotic divisions and its effects suggests that it is connected with the other abnormalities.

The highly reduced numbers of chromosomes in dyads might be the result of several causes. Although somatic cells with chromosome numbers as low as 14, 18 and 20 continue to divide mitotically they are eliminated, probably through competition, by the time microsporogenesis takes place and only the higher numbers occur at this stage (fig. 1B). After first meiotic division, however, an abnormal reduction in number takes place (fig. 1C). This reduction could be the result of spindle abnormalities arising in first division or of lack of association and of co-orientation with resultant lagging. The frequency of the former (table 2) and of laggards and micronuclei (table 5) and the sizes of these micronuclei do not explain this loss however. Stickiness in itself probably contributes in part to this loss, perhaps as a result of fragmentation and disintegration. Thus, for instance, uneven distributions at AI were not observed to be of the order which would result in daughter cells, one of which contained only a small micronucleus as described earlier. Because of the marked stickiness and also the lagging in second divisions, this reduction in chromosome number probably continues when tetrads are formed. Great variation in size of pollen grains (table 6) is therefore to be expected. Under these circumstances a lower percentage of stainable pollen might also be expected.

How then is polyploidy maintained? An explanation may be found in apomixis, which is probably more common in perennial grasses than is suspected. The hybrid shows, however, that pollen grains with chromosome numbers as low as 14 can be produced, and moreover, that such grains are viable and function in crosses.

## 5. BASIC NUMBER

As to the position of this species in a genus with basic chromosome numbers of 7 and 9, several possibilities exist. Either a new basic number for the genus should be added, or another explanation should be found for the occurrence of the number most frequently observed ($2n = 66$). The types of abnormalities in this grass have been found more frequently in hybrids than in species and although 66 is neither

a multiple of 7 nor of 9 the possibility of two such forms producing an allopolyploid is not to be overlooked. It is of course also likely that the peculiar chromosome numbers in this form may have arisen through the more common causes of aneuploidy, regardless of whether the original form was auto- or allopolyploid.

## 6. SUMMARY

1. Chromosome numbers of a *Pennisetum* species vary from 14 to 84 in root tips and 47 to 74 in P.M.C's. Most frequent number in both is 66.

2. Spindle abnormalities and group formation of chromosomes are very frequent in mitosis, less frequent in meiosis. They could be the cause of variation in number.

3. After the first division in the P.M.C. there is an abnormal reduction in chromosome number. This might be attributed to loss which occurs often in both meiotic divisions. The most frequent complement is $n = 23$.

4. A hybrid between this grass and *P. typhoides* ($2n = 14$) with only 21 chromosomes demonstrates the viability of pollen grains with 14 chromosomes.

5. Chromosome numbers in this species indicate (i) that a new basic number should be added to those already known for the genus ($x = 7$ and $x = 9$), or (ii) that it might be a hybrid between $x = 7$ and $x = 9$ forms.

6. The name *P. dubium* is suggested for this species.

*Acknowledgment.*—We are indebted to Professor C. D. Darlington for his suggestions on improving the text.

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# NEW LINKAGE AND INDEPENDENCE DATA FOR RUBY AND JERKER IN THE MOUSE

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## 1. INTRODUCTION

Two comments have recently been made about the linkage relations of ruby (*ru*) and jerker (*je*) of linkage group XII of the house mouse. The earlier (Falconer, 1956) states that these factors "appear to segregate independently of each other in our stocks, though they showed linkage in the original data reported by Fisher and Snell (1948)". The later (Phillips, 1956), which reports a new addition (congenital hydrocephalus, *ch*) to linkage group XIV, records "a slight indication of linkage between *ch* and jerker", and discusses the possibility that groups XIV and XII correspond to one chromosome. As there is doubt about the reality of group XII and thus also about its chromosomal relation with other groups it seems useful to make generally available two further bodies of data. One was obtained some years ago and has until now been published only in a somewhat inaccessible form (Wallace, 1954); the other is newer and has not yet been published at all.

## 2. HISTORY OF NEW DATA

The mice concerned in both the latter bodies of data were bred in this Department and were descended from those used by Fisher in 1948 which provided, with Snell's, the first data on ruby and jerker. Since their genealogical relationship may throw some light upon the paradoxes of the problem a brief history and a summary of their data are first set out.

Fisher and Snell's paper concerned their simultaneous and independent observation of an association between ruby and jerker. The data (table 1) consist of intercrosses and backcrosses both in coupling and in repulsion. It was pointed out that nearly all the separate portions, both in Snell's and in Fisher's summaries favour linkage, and that the total compilation (comprising some 900 mice bred) shows a very significant deviation from 50 per cent. recombination ($\chi^2 = 7{\cdot}22$ for 1 d.f.), with a combined recombination value of about 45 per cent. as derived by Fisher's scoring technique. Group numeral XII, the next available at that time, was assigned to these factors.

Despite the overall homogeneity there was one portion of the Cambridge summary, the repulsion data from segregating males, which did not favour linkage (as may be shown by its negative score

at 50 per cent. recombination ; the value from repulsion males and females together is 52·5 per cent.). The paper mentioned that the repulsion data were the last to be produced (by reason of the derivation of repulsion heterozygotes *via* preparation matings from the coupling), and that more data from repulsion backcrosses were still needed.

TABLE 1

"Linkage" data : *I. After outcross of* ruje *to line 3 in Cambridge 1947. II. After outcross of* ru *to* je *in Bar Harbor 1945*

(Based on the table in Fisher and Snell, 1948)

| Types of mating | I. Cambridge data Phenotypes of progeny | | | | | II. Bar Harbor data Phenotypes of progeny | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | ++ | *ru* | *je* | *ruje* | Total | ++ | *ru* | *je* | *ruje* | Total |
| Intercrosses : | | | | | | | | | | |
| Coupling | 147 | 43 | 53 | 19 | 262 | 4 | 2 | 1 | 1 | 8 |
| Repulsion | — | — | — | — | — | 86 | 35 | 31 | 5 | 157 |
| Backcrosses : | | | | | | | | | | |
| Coupling ♀ | 51 | 30 | 48 | 44 | 173 | 32 | 31 | 30 | 47 | 140 |
| Coupling ♂ | 17 | 15 | 12 | 17 | 61 | 20 | 13 | 13 | 14 | 60 |
| Repulsion ♀ | 4 | 6 | 4 | 4 | 18 | — | — | — | — | — |
| Repulsion ♂ | 5 | 4 | 5 | 8 | 22 | — | — | — | — | — |
| Recombination value | 46·31 per cent. | | | | | 42·17 per cent. | | | | |
| Departure from independence | $\chi^2_1 = 2{\cdot}05$ $0{\cdot}2 > p > 0{\cdot}1$ | | | | | $\chi^2_1 = 6{\cdot}55$ $0{\cdot}02 > p > 0{\cdot}01$ | | | | |
| III. Combined data | | | | | | | | | | |
| Recombination value | 44·74 per cent. | | | | | | | | | |
| Departure from independence | $\chi^2_1 = 7{\cdot}22$ $0{\cdot}01 > p > 0{\cdot}001$ | | | | | | | | | |
| Heterogeneity | $\chi^2_1 = 1{\cdot}37$ $0{\cdot}3 > p > 0{\cdot}2$ | | | | | | | | | |

Further work on the association of ruby and jerker continued in this Department until the present time.

Fisher's 1948 data concerned the segregation in 1947 of these factors immediately after their introduction to Line 3 of the 21 Segregating Inbred Lines, where they were combined with other factors. (These Lines had been set up by Fisher [1949*a*] to discern new linkages and to accumulate the large bodies of data needed to confirm—or to contradict—indications of loose linkage.) It is clear then that the data were from relatively *outbred* material : in fact there are only three sib matings and these are non-successive. In accordance with the strict sib mating policy subsequently followed in the Lines the next body of data is on the other hand very *inbred* (table 2).

This concerns the matings made up from January 1948 until July 1952 by Miss M. F. I. Speyer who became responsible for Line 3. They cover some eight generations of inbreeding. It is not as large

a body of data as the earlier one (some 640 mice were bred), but as it consists solely of backcrosses, it is equally informative—about 2600 units of Information. Although repulsion data are still in the minority this phase is now sufficiently well represented to balance the coupling. The striking feature here is that in contrast to the earlier data both phases fail to show linkage.

A little further data accumulated during the following year (1952-3), but it is not reported here because the ruby : normal ratio is heterogeneous as between matings and there is a serious depletion of jerkers. The Line became impossible to continue, more and more of the matings being infertile, and in July 1953 it had virtually died out.

TABLE 2

"Independence" data: *from eight generations of inbreeding after the data for table 1 were obtained. Cambridge 1948-52*

| Types of mating | Phenotypes of progeny | | | | | Recombination value | Departure from independence |
|---|---|---|---|---|---|---|---|
| | ++ | ■ | *je* | *ruje* | Total | | |
| Backcrosses : | | | | | | | |
| Coupling ♀ . | 62 | 59 | 51 | 51 | 223 | 52·58 per cent. | $\chi^2_1 = 0{\cdot}98$ |
| Coupling ♂ . | 53 | 63 | 49 | 45 | 210 | | |
| Repulsion ♀ . | 17 | 20 | 15 | 27 | 79 | | $0{\cdot}5 > p > 0{\cdot}3$ |
| Repulsion ♂ . | 31 | 27 | 34 | 35 | 127 | | |

The figures here are slightly smaller than those given earlier (Wallace, 1954) because the earlier compilation included in error some data from the outbred material.

To preserve the combination of factors in the Line, two outcrosses were then made to another inbred stock, and from their descendants a new Line was formed by Mrs M. A. C. MacNeil. The first matings segregating for ruby and jerker were intercrosses. From these and their descendants came known repulsion backcrosses and backcrosses whose linkage phase was unknown ; and from these in turn came known coupling backcrosses. Their pedigree covers about six generations from the outcross, the last mating breeding early in 1956. While each generation was more inbred than the previous one, only the last three were sib mated. The material is similar in genealogical origin to that of Fisher, being relatively outbred, and it is in strong contrast with the 1948-52 material which was much more inbred.

Table 3 summarises the known coupling and repulsion data. The intercrosses are omitted because the ruby : non-ruby and jerker : non-jerker ratios are seriously disturbed ; and the backcrosses of uncertain phase are also omitted, for obvious reasons. In the remaining data, ruby is somewhat inviable but as the jerker ratios do not deviate significantly from expectation, there is no disturbance to linkage and the usual process of scoring is a sufficiently accurate way of determining the amount and significance of deviation from independent assortment.

The important feature here is that, as in the 1947 data, and in contrast to the 1948-52, linkage is favoured. Of the three, this is the smallest body of data (some 81 mice were bred, giving 339 units of Information). The estimated linkage value is close to the 1947 one, about 40 per cent. The repulsion data, as in the 1947, show weaker linkage than do the coupling and it is unfortunate that no more data are available.

In 1956, before the reappearance of linkage had been noticed, Line 3 was terminated and the factors contained in it used elsewhere.

TABLE 3

"Linkage" data: *from matings following an outcross. The outcross was made after the data in table 2 were obtained. Cambridge 1954-56*

| Types of mating | Phenotypes of progeny | | | | | Recombination value | Departure from independence |
|---|---|---|---|---|---|---|---|
| | ++ | *ru* | *je* | *ruje* | Total | | |
| Backcrosses: | | | | | | | |
| Coupling ♀ . | 15 | 5 | 11 | 9 | 40 | 39·53 per cent. | $\chi^2_1 = 3{\cdot}41$ |
| Coupling ♂ . | 4 | 1 | 2 | 2 | 9 | | |
| Repulsion ♀ . | 11 | 1 | 11 | 5 | 32 | | $0{\cdot}1 > p > 0{\cdot}05$ |

## 3. THE DATA FROM EDINBURGH

Mice carrying ruby and jerker, derived from Fisher's 1947 material, were sent to Dr Falconer and used to obtain further data (1956). He has kindly supplied me with a compilation and the following information. All the material is outbred. Crosses were made of ruby jerker animals to normals from eight unrelated sources; $F_1$ from three of the outcrosses were intercrossed, providing coupling intercross data, and $F_1$ females from six of the outcrosses were backcrossed to males of the ruby jerker stock, providing coupling backcross data (table 4). The recombination value from the intercrosses is estimated as 48·1067 per cent. and that from the backcrosses as 51·3803 per cent. The combined value of $50{\cdot}9713 \pm 1{\cdot}62$ per cent. has 3811 units of Information, slightly more than were given by any of the earlier bodies of data.

A closer analysis of the more informative material, the backcrosses, reveals no significant heterogeneity in the ratio of non-recombinants to recombinants between the groups of matings from the six sources ($p = 0{\cdot}5$ to $0{\cdot}3$). There is, however, a discrepant ratio, 16 : 33, from females derived from one outcross mating: it gives a recombination value of 67·3 per cent. and a $\chi^2$ of 5·8980 for 1 d.f.; but no such anomaly is seen in the females from a second outcross to the same stock, and this single observation is not sufficiently large to produce overall heterogeneity. The intercross data also, when grouped according to their three sources, appear to be homogeneous.

## 4. ANALYSIS OF THE TOTAL DATA

It should perhaps be noted that the single-factor ratios in all the bodies of data now given conform to expectation (except in the Cambridge 1954-56 data where only one of them is disturbed). There is therefore no question of bias in the estimates of linkage due to viability disturbance. If there were, it would be desirable to have about equal amounts of coupling and repulsion data, at least in the backcrosses : in fact, there are very little repulsion data. Although this unbalance is not important from the point of view of linkage estimates, it is a regrettable weakness in the foundation upon which hypotheses other than linkage may have to be erected.

Clearly the data are not consistent in the linkage values they yield.

TABLE 4

" Independence " data : *from matings following outcrosses of* ruje *mice derived from Cambridge in 1947. Edinburgh 1956*

| Types of mating | Phenotypes of progeny | | | | | Recombination value | Departure from independence |
|---|---|---|---|---|---|---|---|
| | ++ | *ru* | *je* | *ruje* | Total | | |
| Intercrosses : Coupling . . | 158 | 53 | 44 | 17 | 272 | 50·97 per cent. | $\chi^2_1 = 0{\cdot}04$ |
| Backcrosses : Coupling ♀ . | 209 | 224 | 204 | 196 | 833 | | $0{\cdot}9 > p > 0{\cdot}8$ |

It may be supposed that environmental factors such as temperature and diet are the cause of fluctuation in chiasmata frequency between the two loci. This possibility cannot easily be excluded, for even within one laboratory there may be unsuspected changes over a long period of time, but it is safe to say that there have been no obvious ones in Cambridge, yet the Cambridge data are significantly heterogeneous. This is apparent from the following calculation. If the estimated recombination values for the 1947, the 1948-52 and the 1954-56 material are designated $\hat{\theta}_1$, $\hat{\theta}_2$ and $\hat{\theta}_3$ respectively, with Information parameters $I_1$, $I_2$ and $I_3$ respectively, a combined estimate, $\hat{\theta}$, may be obtained from the formula

$$\hat{\theta} = \frac{I_1\hat{\theta}_1 + I_2\hat{\theta}_2 + I_3\hat{\theta}_3}{I_1 + I_2 + I_3}.$$

This is 49·0476 per cent. The formula gives a weighted mean to $\hat{\theta}$ and leads to the $\chi^2$ formula

$$\chi_2^2 = I_1\hat{\theta}_1^2 + I_2\hat{\theta}_2^2 + I_3\hat{\theta}_3^2 - (I_1 + I_2 + I_3)\hat{\theta}^2$$

which is thus an appropriate test of heterogeneity. At 6·3488 for 2 d.f. it is significant, $p$ being between 0·05 and 0·02.

It is interesting that the combined $\hat{\theta}$ is virtually 50 per cent. Further, when the Bar Harbor 1947 and Edinburgh 1956 values are

included in the formula (see below), the combined $\hat{\theta}$ remains at almost exactly the same value, 49·0470±1·4739 per cent.

| Cambridge | | | Bar Harbor | Edinburgh |
|---|---|---|---|---|
| 1947 | 1948-52 | 1954-56 | 1947 | 1956 |
| $46{\cdot}4310 = \hat{\theta}_1$ | $51{\cdot}9561 = \hat{\theta}_2$ | $39{\cdot}5259 = \hat{\theta}_3$ | $42{\cdot}1700 = \hat{\theta}_4$ | $50{\cdot}9713 = \hat{\theta}_5$ |
| $1606{\cdot}426 = I_1$ | $2556{\cdot}09 = I_2$ | $339{\cdot}3432 = I_3$ | $1066{\cdot}68 = I_4$ | $3810{\cdot}60332 = I_5$ |

These values used in the $\chi^2$ formula, give even more significant heterogeneity, for $\chi^2$ for 4 d.f. is 12·8023 with $p$ between 0·02 and 0·01.

## 5. DISCUSSION

It is perhaps a platitude that the results of later work do not diminish the significance of the earlier. It is, however, sometimes salutary to restate it, especially in conjunction with a further one, namely that the establishment of independence is not proof of localisation on different chromosomes. The reality of linkage group XII is not disproved, but the manifest heterogeneity within the data requires an explanation in terms of linkage ; or, if this is not found possible, a search must be made for other hypotheses and a discriminating experiment designed.

The possibility of environmental disturbance to linkage has been mentioned. Such factors as temperature and age are well-known to affect linkage values in *Drosophila*. The absence of records of such effects in mice is probably due to the general lack of sufficiently accurate linkage data in which they may be discerned. There are two experiments where the design envisaged this kind of analysis and where an age effect was found (Fisher, 1949*b*, and Wallace, 1957), and it is now recognised that if experimental work allows of its observation, sex differences in recombination are very commonly found. It is conceivable that loose linkages, particularly those that span the centromere, are very susceptible to these and to other as yet unknown environmental agents.

That this may be true of genetic agents is not so evident : for there are no recorded instances in mice—as there are in *Drosophila* and other organisms—of spontaneous translocation and inversion, or of mutations specifically increasing or decreasing the amount of recombination. This again may be due to the lack of careful experimentation. The presence of such phenomena in other organisms should suggest, until it is proved otherwise, that their existence is possible in mice. Indeed, heterogeneous data provide the first kind of evidence which should be sought, and it is unfortunate that there is probably a tendency to withhold such data from publication because the cause of heterogeneity

has not been investigated. Finally, artificial selection has been known to decrease and to increase recombination in certain organisms (*e.g. Matthiola*, see Fisher, 1933) ; moreover, inbreeding has also been known to change the properties of chiasma formation, usually reducing the frequency (Rees and Thompson, 1956). These observations point to the existence of minor genetic agents having a cumulative effect ; the lack of experimental work on this point in mice leaves open the possibility of their existence.

The present data do not allow of the isolation of any one of these possible causes of heterogeneity. However, it may be noted that there are two striking features which throw doubt on the hypothesis of linkage itself. First, the data as a whole favour independence. Secondly, the three * sections which significantly contradict independence arose from outbred material : this conflicts with the view that the lower linkage values are due to the fortuitous accumulation of genetic factors tending to tighten linkage, for these are more likely to be dispersed by outbreeding than to be maintained by it.

Here again, it is perhaps useful to restate a further platitude : that such dispersion is expected because outcrossing promotes heterozygosity and breeding from outcrossed material produces segregation. For this suggests an alternative hypothesis : affinity (Michie, 1953, and Wallace, 1953). Outcrossing may be expected to result in heterocentricity and inbreeding in homocentricity. To state this more precisely : Quasi-linkage of two factors may be supposed to be due to their proximity to centromeres on different chromosomes, the centromeres being capable of showing preferential association at meiosis. Inbreeding is always expected to result in homocentricity ; in this case, quasi-linkage disappears. Outbreeding is expected to result *either* in homocentricity *or* in heterocentricity ; if heterocentricity results, quasi-linkage reappears. Outbreeding will result in homocentricity if the two stocks concerned in the outcross have the same centrotypes for the chromosomes marked by the two factors, and it will result in heterocentricity if they have different centrotypes. These and related points have been explained fully in a recent paper (Wallace, 1958, p. 217-18). It is sufficient to notice here that none of the present bodies of data conflict with these expectations : for the inbred section shows independence, three of the outbred sections show a linkage-like association and one of them shows independence.

Closer examination of these bodies of data reveals a closer fit to expectation on an affinity hypothesis. Firstly, the repulsion data in the Cambridge 1947 compilation is from slightly more inbred material than the coupling ; the weakness of the linkage it shows may be interpreted as a result of the inbreeding. Secondly, the Bar Harbor coupling matings are slightly more inbred than the repulsion intercrosses (private communication), and give data showing

* These are the Cambridge 1947 and 1954-56, and Bar Harbor 1945 data. That the latter are outbred is confirmed by Snell (private communication).

weaker linkage than the repulsion (see table 1 : the recombination values are about 43 and 37 per cent. respectively).

Thirdly, a heterogeneity test of the Cambridge 1948-52 inbred data, while formally exhibiting homogeneity ($p$ is 0·2 to 0·1), discloses the fact that three of the twenty-three $\chi^2$ values contributing to the total $\chi^2$ have probabilities less than 0·05 : these are $\chi^2$ values of 4·7647, 5·5556, and 7·7586. Some degree of heterogeneity is not unexpected since this stock represents progressive inbreeding and not all matings can be expected to be homocentric at once : that is, the stock should be expected to consist mainly of matings showing independence and partly of matings showing deviations from 50 per cent. In fact, the segregation giving $\chi^2 = 4{\cdot}7647$ gives a value less than 50 per cent., and the other two a value exceeding 50 per cent. A linkage hypothesis cannot tolerate for the same factors values both less than and greater than 50 per cent., but an affinity one can. (Segregations giving values exceeding 50 per cent., *i.e.* " reversals ", arise from heterozygotes in which the maternal centromere of one pair of homologous chromosomes associates at meiosis with the paternal centromere of the other pair of homologues and vice versa.)

Finally, the Edinburgh 1956 outbred material (which showed independence) reveals a similar situation. As has been stated, eight different stocks were used in the outcrosses. It is reasonable to expect that by chance some of these should have resulted in quasi-linkage and others not, whereas the apparent homogeneity of the data ($p = 0{\cdot}5$ to 0·3) suggests that there were no quasi-linkages. However there is one significantly discrepant segregation (that giving a recombination value of 67·3 per cent.), and this does at least suggest that the outcross stock concerned was of a different centrotype from the ruby jerker stock. The second outcross to it did not reproduce the quasi-linkage, but this may have been due to heterocentricity in one or both chromosomes in the ruby jerker stock (which was not closely inbred).

The interpretation to be made of the results of these heterogeneity tests depends on the meaning of " formal significance ". Perhaps a fourth platitude may be excused here on account of its relevance. Lack of significance at the 5 per cent. level does not necessarily disprove heterogeneity—the progeny numbers in the individual matings and sets of matings, and the occasional deviations from 50 per cent. due to quasi-linkage, may both be too small to produce a heterogeneity $\chi^2$ significant at that level. In the present case this is very likely so, for the recombination values for ruby and jerker so far encountered have been, in general, very loose (40 per cent. or higher), and the progeny numbers from each set of matings (Edinburgh, 1956) average 119 and from each mating (Cambridge, 1948-52) average 24. It is therefore probably true to say that the amount of heterogeneity exhibited by these two bodies of data is about as much as should be expected.

To sum up : Quasi-linkage is expected to occur in some, but not

necessarily in all, outcross bodies of data, and to be absent in inbred ones. Both expectations are fulfilled. Further, the results of internal analysis definitely favour affinity rather than linkage. However, linkage is not an untenable hypothesis because the existence of factors capable of causing fluctuation in the recombination value cannot be excluded.

Evidence which may be sought experimentally to distinguish between these two phenomena is of several kinds. For example, a further outcrossing programme might result in the renewed observation of values significantly exceeding 50 per cent. If any of this material showed linkage values less than 50 per cent. it could be manipulated so as to produce (if affinity were operating) animals capable of giving values exceeding 50 per cent. Further repulsion data could also be produced and would be informative. Tests of linkage of ruby and jerker, simultaneously with factors in other linkage groups and thus likely to be on other chromosomes, might result in the establishment of non-linear linkage relations between them : according to what these were, it might then be possible to conclude either that ruby and jerker are linked and to locate the centromere in relation to them, or that they are not linked and to define the maximum distance of each from its own centromere. (These ideas have been developed elsewhere : Wallace, 1958). Unfortunately, although one of Phillips' experiments with congenital hydrocephalus did involve ruby and jerker simultaneously (private communication), her data show severe viability disturbance and the estimated recombination values are insignificantly below 50 per cent., so that no realistic conclusion can be drawn.

It is possible that other cases of heterogeneous linkage data may be explained on the basis of affinity. One case of non-linearity of the linkage relations between three markers has been thoroughly investigated and shown to be fully explicable on this basis (Wallace, 1958). The disappearance of an association on inbreeding, the observation of anomalous linkage relations, and the discovery of heterogeneity are likely to have led to the abandonment of data without publication. It would be of considerable interest if such cases were published and others searched for in past records.

## 6. SUMMARY

Two new bodies of data from Cambridge on the association of ruby and jerker in the mouse are made available. One shows independent segregation and the other linkage. All the evidence on record is reviewed and discussed. Interpretations based on linkage and on affinity are given, and the design of discriminating experiments sketched.

*Acknowledgments.*—I am indebted to Sir Ronald Fisher, Miss M. F. I. Speyer and Mrs M. A. C. MacNeil for permission to use the data in this Department

(hitherto unpublished) and to Dr D. S. Falconer for permission to analyse the data published briefly in *Mouse News Letter*.

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# A NEW GENE FOR DEAFNESS IN THE MOUSE

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Two years ago Deol (1956) reported the first case of inherited deafness in the mouse not associated with any of the usual concomitant locomotor disturbances. It was followed by a systematic search of the mouse colony of this laboratory, which resulted in the discovery of several other cases of the same kind. The mutant gene described here is one of them. The name *deafness*, symbol *dn*, is proposed for it.

## 1. HEARING ABILITY AND BEHAVIOUR

Hearing ability was tested by means of a click produced by striking a pair of forceps on a bottle. Refined methods were considered unnecessary as deafness could be recognised by this test without ambiguity in adult animals. Normal mice respond to it with a general muscular twitch which is most noticeable in the ear pinna. No such response was ever obtained in mice homozygous for *deafness*. The heterozygotes on the other hand were always normal, and the onset of hearing took place between 10 and 14 days depending on the size, general fitness and, perhaps, the genetic constitution of the individual. A number of the heterozygotes were kept until they were well over a year old and their hearing ability was found to be unimpaired.

Though in general the behaviour of *dn*/*dn* is normal, tossing movements of the head have been observed in some individuals. This is a barely perceptible abnormality and can be best described as an accentuation of the normal sniffling movements. But it is quite distinct from the latter, especially to an observer familiar with the mutants of the shaker-waltzer type.

## 2. GENETICS

The mutant gene *dn* was discovered in the curly-tail stock of this laboratory, its origin being independent of the *ct* gene. It must have been in that stock for some time as it was already fairly widely spread when first detected. The segregation data of table 1 show a close approximation to the ratios expected, if *dn* is a recessive gene with regular manifestation.

It seems that the presence or absence of head tossing (HT) is influenced by the residual genotype. The data of table 2 show that, in matings between two *dn*/*dn* animals, the incidence of head tossing is lowest when neither parent, and highest when both parents toss their heads ($\chi^2 = 8{\cdot}511$ ; $n = 2$ ; $P = 0{\cdot}015$).

The double heterozygotes

+/*dn* ; +/*df*
+/*dn* ; +/*sh*-1
+/*dn* ; +/*sh*-2
+/*dn* ; +/*v*
+/*dn* ; +/*je*
+/*dn* ; +/*pi*

all show normal behaviour and can hear. In the case of *dn* and *df*, this has been followed to the age of 8 months, in the other cases for

TABLE 1

*Segregation of* dn

| Mating | Normal | Deaf | Total |
|---|---|---|---|
| +/+ × *dn*/*dn* | 33 | ... | 33 |
| *dn*/*dn* × *dn*/*dn* | ... | 218 | 218 |
| +/*dn* × +/*dn* | 144 | 57 | 201 |
| +/*dn* × *dn*/*dn* | 61 | 61 | 122 |

TABLE 2

*Incidence of head tossing* (*HT*) *in* dn/dn *animals*

| Mating | Normal (N) | Head tossing (HT) | *n* | Per cent. HT |
|---|---|---|---|---|
| N × N | 52 | 2 | 54 | 3·7 |
| N × HT | 16 | 3 | 19 | 15·8 |
| HT × HT | 12 | 5 | 17 | 29·4 |

shorter periods. It thus appears that *dn* is not an allel of any of these genes. A test of *dn* against *Va* has not yet been carried out. A test of *dn* against the genes for *kr*, *dr* and *fi* was not considered necessary in view of the totally different pathological situation.

## 3. HISTOLOGY

The same technique was used as has been described by Deol (1954). It must be mentioned here that the embedding time for 56° C. paraffin was given only vaguely in that publication. It should be put at one hour. If harder paraffins are to be used, the embedding time must presumably be even more reduced (say 45 minutes for 60° C. paraffin).

Serial sections of the labyrinths of 35 mice were cut, some at $7\frac{1}{2}\ \mu$ and some at $10\ \mu$. The age of the mice ranged from 9 to 388 days (one-day-intervals between 9-15-day stages, longer intervals later on). Only a few of the normal controls were taken from the deafness stock ;

the rest came from previous investigations. In addition series of the sacculi of normal and head-tossing animals, about one year old, were cut (see table 3).

The pathological changes observed in the cochlea of *dn*/*dn* mice closely resemble those of *df*/*df* mice and indeed those of the degenerative group of mutants of the shaker-waltzer type (waltzer, shaker-1, shaker-2, jerker, pirouette and varitint-waddler). The earliest abnormalities of the organ of Corti appear at the age of 10 days when Deiters's cells begin to lose their identity: first their boundaries become indistinct and then the nuclei move to different levels. First changes in the hair cells can be clearly seen on the 15th day. Abnormalities of the stria vascularis can be detected from the 10th day

TABLE 3

*Histological anomalies of the maculæ sacculi*

| Head tossing | | Normal behaviour | |
|---|---|---|---|
| Right ear | Left ear | Right ear | Left ear |
| ++ | ++ | ... | + |
| – | ++ | – | ... |
| – | ++ | ++ | + |
| ++ | ... | (+) | (+) |
| ++ | +++ | ... | – |
| ++ | ... | + | ++ |
| (+) | + | (+) | (+)? |
| – | ... | – | – |

onwards. The free end of the tectorial membrane does not lie over the hair cells but curls up. When a *dn*/*dn* mouse is about three weeks old most of the elements of the scala media complex are well on their way to degeneration, but the degeneration of the spiral ganglion does not seem to begin until about 50 days. This may be due to the difficulties involved in detecting any but gross pathological changes in the ganglion cells. In old mice, that is, 9 months or over, the degeneration of Corti's organ as well as the spiral ganglion is nearly complete. At this stage Corti's organ is a mass of dedifferentiated cells though the tunnel of Corti does not usually collapse (sometimes one or both rods may be missing), and the spiral ganglion has only a few remnants of the nerve cells left.

Degeneration of the macula of the sacculus was observed in head-tossing as well as in normally behaving *dn*/*dn* mice. In general it is of the same kind as is found in the shaker-waltzer group (Deol, 1954). Table 3 shows the degree of degeneration of the available maculæ sacculi. Normal maculæ are denoted by –; (+) means that only a few hair cells are histologically abnormal. In severe cases (indicated ++) the hair cells degenerate completely and the otolith membrane

comes to rest on the supporting cells. In some mice the otolith membrane may degenerate and the otoliths be lost. In one case (shown by +++) virtually the whole macula has disappeared.

Histological anomalies of the maculæ sacculi thus occur both in head-tossing and in normally behaving *dn*/*dn* mice though the former perhaps tend to be more strongly affected. Obviously, slight anomalies of the macula are compatible with normal behaviour. Whether more extreme lesions are responsible for head tossing must remain an open question.

## 4. SUMMARY

1. A new recessive gene for uncomplicated deafness has been discovered ; the name *deafness*, symbol *dn*, is proposed for it.

2. As judged by a simple test, *dn*/*dn* mice cannot hear at any time of their life while the hearing ability of the heterozygotes is unaffected.

3. Slight head tossing occurs in some *dn*/*dn* mice. Its incidence is influenced by the residual genotype.

4. Beginning about the 10th day, degeneration of the cochlear elements occurs in the homozygotes.

5. Degeneration of the macula of the sacculus may occur both in head tossing and in normally behaving mice, though some ears remain histologically normal even in old animals. The time of onset of the macular degeneration is not known.

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# THE PARTITION OF DOMINANT LETHALS IN THE MOUSE BETWEEN UNIMPLANTED EGGS AND DECIDUOMATA

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## 1. INTRODUCTION

MATINGS of males during the first few weeks after X-radiation yield litters of reduced size. It has been repeatedly demonstrated that this reduction is due to two phenomena : fertilised eggs which fail to evoke an implantation reaction, and implantations which show no sign of a fœtus. It is not yet known whether the embryos which invoke the deciduomatal reaction are dead at the time they exert their stimulus. If not, they must die very soon afterwards.

According to Brenneke (1937), the proportion of cytologically abnormal eggs increases as cleavage proceeds. Some eggs showed micronuclei at the 2-celled stage, but a higher proportion showed them in 3- to 6-celled stages. She also found that the proportion of implantations was greater than the proportion of normal cleavages in parallel matings, which has been interpreted by Russell (1954) to mean that some eggs showing abnormal cleavages are quite capable of invoking a deciduoma.

Lea (1947) suggested that the larger the acentric fragment lost as a result of a single break, and the greater the number of breaks, the earlier would the embryo be lost. Russell *et al.* (1954) have observed that " the relative percentages of death occurring before and after implantation vary with dose ".

The above account summarises about all that is known of the causes of the striking dimorphism in phenotype of dominant lethals as observed in rodents. It is the purpose of this paper to investigate this dimorphism further. Though, theoretically, dominant lethals could be responsible for any deaths from implantation to parturition (and even much later), these phenotypes are not considered here because, since their incidence from implantation at $4\frac{1}{2}$ days to dissection at $12\frac{1}{2}$ days is unaffected by dose (Bateman, 1958), they would appear to have an insignificant dominant lethal component.

## 2. THEORETICAL

Semi-sterility resulting from heterozygosity for translocations provides evidence relevant to the consideration of dominant lethals, for it has as its immediate cause the death of approximately half the embryos. These lethal embryos have one normal chromosome set from the normal parent, while the other set carries both a duplication

and a deficiency as a result of recombination between the original and the reciprocally translocated pairs of chromosomes in the semi-sterile parent. Thus the duplication-deficiency behaves as a dominant lethal : it is a *synthetic dominant lethal*.

The extensive data of Carter *et al.* (1955) show that the dominant lethals are nearly always expressed as deciduomata (abbreviated in the rest of this paper to ED for " Early post-implantation Death "). Only one (viz. T8) out of eight translocations shows an appreciable effect on pre-implantation losses (henceforward abbreviated to PRE), which represented 10 per cent. of the zygotes, and in this case the zygotic loss was more like 60 per cent. than 50 per cent. Such an excess would be expected if a proportion of the quadrivalents at meiosis showed " Adjacent II " separation at first anaphase.* While other kinds of numerical disjunction give only 50 per cent. unbalanced gametes and these having chromosome *segments* duplicated and deficient, Adjacent II gives only 100 per cent. unbalanced gametes, and half of these have *whole* chromosomes duplicated and deficient. The genetic unbalance of the resulting zygotes will, on the average, be twice as severe, though the relative severity with any given translocation will depend of course on the precise points of exchange. It is a likely hypothesis that the 10 per cent. PRE in T8 corresponds to embryos carrying the extreme duplications and deficiencies arising from half the gametes produced by Adjacent II separation at meiosis. This argument has been based on the assumption that, in the mouse, all chromosomes are effectively telocentric.

The implication of the data from semi-sterile matings is thus that unbalance of the order of a single duplication of one chromosome segment and a single deficiency of another produces ED, whilst the more severe unbalance corresponding to a duplication and deficiency of whole chromosomes may lead to PRE. It may be worth pointing out here that neither ED nor PRE in semi-sterility will be associated with micronuclei, which are therefore not the cause of death but merely an indication of chromosome breakage within the last few cell generations.

Let us now consider *primary* dominant lethals. Owing to their different origin they are of rather different genotypes. Single unrestituted breaks induced in sperm will result in the loss of the acentric fragment at the first cleavage division, with the production of a micronucleus. Unless one postulates the healing of centric fragments, these will be expected to form a dicentric by sister reunion, and form a micronucleus, or be wholly included in one blastomere and absent from the other, or start a breakage-fusion cycle inevitably leading to its elimination, or, finally, the telophase bridge at first cleavage may

* When adjacent centromeres on a ring quadrivalent go to the same pole, though there is *numerical* disjunction there may be non-disjunction of homologous centromeres. McClintock has called this " Adjacent II " anaphase separation to distinguish it from anaphase separation in which disjunction of homologous centromeres is combined with adjacent centromeres going to the same pole (" Adjacent I ").

persist and interfere with subsequent divisions. In any event the broken chromosome will be completely lost. Lea's hypothesis (1947) that the severity of a dominant lethal will depend on the size of the acentric fragment would then be fallacious. The severity would depend on the original chromosome broken.

Theoretically, at least, one can distinguish between *mechanical* lethals where the mitotic mechanism is interfered with and *genetical* ones directly attributable to the loss of whole chromosomes and the genes borne on them. Mechanical lethals might be expected to take effect earlier than genetical ones as they would interfere with cleavage, whereas the absence of a chromosome would be more likely to express itself when differentiation began (cf. semi-sterility, *supra*, where all lethals are of the genetical type).

Seidel (1952) has demonstrated that a single blastomere from a 2-celled stage is fully capable of developing into a normal embryo. Therefore fractional dominant lethals, that is those affecting only some of the products of division of the treated sperm, would probably be undetectable, at least if one of the first cleavage nuclei was unscathed.

Lea's second hypothesis, that the time of death of a zygote was determined by the number of breaks it carried, seems very reasonable.

The number of X-ray induced dominant lethals per egg would be expected to be distributed in a Poisson series with a mean, $\mu$, proportional to dose or sensitivity. The first term (zero lethals) would correspond to the live fœtuses. Let us assume tentatively that embryos with a single lethal survive to implantation, and all with more than one die unimplanted. The frequencies of live fœtuses (IMP—ED), ED and PRE as proportions of all corpora lutea (CL: total of corpora lutea or ova ; IMP : total number of implantations) will be $e^{-\mu}$, $\mu e^{-\mu}$, and $1-(1+\mu)e^{-\mu}$ respectively. (Hypothesis A.) It follows very conveniently that the ratio ED/(IMP—ED) is equal to $\mu$.

Hypothesis A predicts the relation between ED/CL and PRE/CL. As the gross mutation rate rises from zero to about 0·25 it will be expected to consist almost entirely of ED/CL. With further rise the contribution to PRE/CL will become manifest and increase with ED/CL up to a gross rate of about 0·6, when PRE/CL will continue to increase at the expense of ED/CL. This expectation is shown in figs. 1 and 2, after allowance for the control value of PRE/CL (0·1) which is supposed to arise from some cause other than mutation. The crudest alternative hypothesis, that ED and PRE comprise constant fractions of dominant lethals, would produce a straight line as shown in the figs. (Hypothesis B.) As a compromise, Hypothesis C assumes that not only all single-lethal embryos survive to implantation, but also one quarter of multi-lethal embryos similarly survive to that stage. This proportion is an *ad hoc* value chosen to suit the actual observations shown in the figs. The earlier discussion of genetic and mechanical lethals provides the basis for a fourth hypothesis : mechanical lethals contribute only to

PRE, whereas genetical lethals, unless very severe, will contribute only to ED. Single breaks would cause the eventual loss of *single chromosomes* and therefore be expected to produce ED (see above). Plural breaks not involved in rearrangements would cause the loss of more than one chromosome and be more likely to produce PRE with increasing numbers of chromosomes lost. Balanced rearrangements would be viable. Unbalanced rearrangements would usually, but not invariably, depending on the relationship of the points of exchange to the centromeres, cause anaphase bridges. Consequently they might be the main source of mechanical lethals (PRE), though

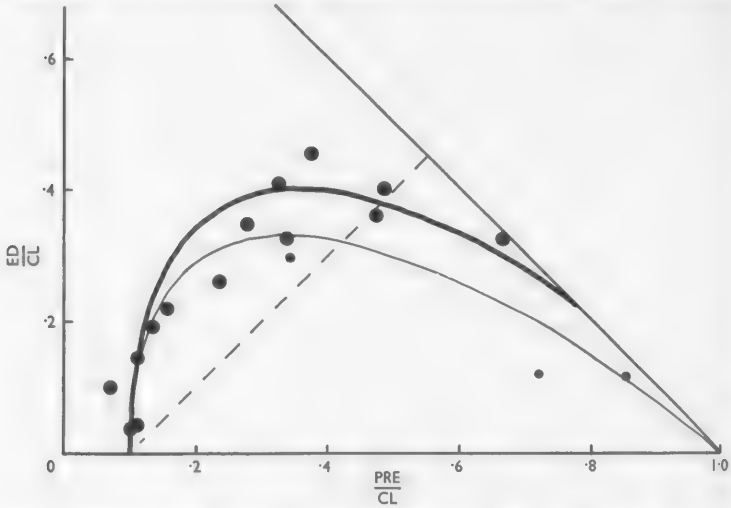


FIG. 1.—Relationship of ED/CL to PRE/CL for author's X-ray data. Dots: observed values, small dots being based on < 100 CL. Thin entire curve: expectation on hypothesis A. Broken line: expectation on hypothesis B. Thick line: expectation on hypothesis C. The diagonal line shows the limiting value: ED/CL+PRE/CL = 7.

some without bridges would act as genetic lethals (ED). The overall results would be empirically much the same as under Hypothesis C.

## 3. THE DATA

The first three hypotheses are compared with my own data in fig. 1. Only matings within three weeks of irradiation have been included as there is good evidence that the rate of fertilisation varies after that time. The dose varied from 125 r to 2000 r. Control values are also shown. Most of the data have been already published (Bateman, 1958). The sum of the $X$ and $Y$ ordinates gives the total incidence of dominant lethal embryos. The diagonal therefore represents the limit of any curve ($x+y = 1$).

Hypothesis B is obviously unsuitable. A and C are equally suitable

up to an ED/CL rate of 0·3. The very slow initial increase in PRE (it is still indistinguishable from the control level for the third week after 125 r and the first two weeks after 200 r) is strong evidence for a Poisson distribution of the number of dominant lethals per egg, combined with the late survival of embryos with a single lethal. At the higher levels of lethal incidence, however, it would appear that C is superior to A in that many embryos with more than one lethal are capable of surviving to implantation.

Russell *et al.* (1954) have an extensive series of data following the exposure of male mice to neutrons. Their series is compared with

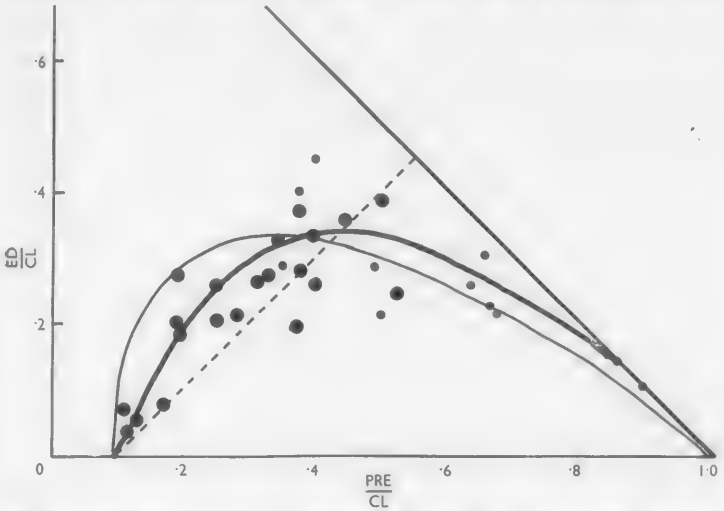


FIG. 2.—Relationship of ED/CL to PRE/CL for Russell's *et al.* neutron data. Key as in fig. 1 *except that* Thick line : expectation on hypothesis C, modified empirically to allow for the clustering of neutron-induced breaks.

the same hypotheses in fig. 2. A rather different picture is presented. Though there is the same decrease in ED at the highest gross mutation rates, there is now a strong correlation between ED and PRE at the lower rates, with PRE seeming to comprise about a third of the lethals. This is compatible with what is known of the genetic effects of neutrons, namely that a single neutron track can induce more than one break per nucleus (Giles, 1943, and Muller, 1954). The number of breaks per nucleus will therefore no longer follow a Poisson distribution, which only applies to independent events. The data imply that about a third of the neutron tracks producing lethals produce more than one per nucleus. However, as the number of effective tracks per nucleus rises the proportion of ED is bound to fall as in Hypothesis A or C. The empirical curve has been drawn freehand to represent a situation approaching B at low gross incidence of lethals and changing over to C at higher incidence.

Summarising, then, both sets of data are consistent with the theory that eggs with a single chromosome break produce deciduomata, and that most eggs with more than one break are lost before implantation. This will be partly due to the greater severity of an effect of the same nature as that found with single lethals, but it might also be due to aneucentric rearrangements causing mechanical breakdown of cleavage.

## 4. MEASUREMENT OF MUTATION RATE

As stated earlier, *if* lethals were distributed between eggs in a Poisson series and ED represented exclusively eggs with single lethals, the best estimate of mutation would be ED/(IMP−ED), (*a*). We know now that these premisses are not strictly true, even for X-rays. It becomes necessary therefore to consider alternative estimates. One can derive a function from the total of lethal eggs. This is simply the negative of the natural logarithm of the proportion of fertilised eggs which survive. It is necessary first to know the proportion of unfertilised eggs. It would appear from control data and from the first three weeks after irradiation (see figs. 1 and 2) that this runs at a constant value of 10 per cent., but in later weeks it is variable and often much higher. When the fertilisation rate is known to be 90 per cent. it can be allowed for, to produce the following parameter :

$$-ln\left(\frac{1}{0{\cdot}9}-\frac{\text{PRE}+\text{ED}}{0{\cdot}9\times\text{CL}}\right)\ (b).$$

As the number of lethals per egg rises, there is a saturation effect so that the proportion of affected eggs does not rise linearly with mutation rate but as a negative exponential with a maximum of unity. Function *b* corrects this and increases linearly with mutation rate. When, however, the rate of fertilisation is unknown, this function is useless. A third function bears an intermediate relationship between *a* and *b*. It has the same principle as *b* but has the advantage of being independent of fertilisation rate. It is $-ln(1-\text{ED}/\text{IMP})$, (*c*).

Unfortunately there is no absolute standard of mutation rate against which these three functions can be compared. One can, however, compare them with each other according to their dose-dependence. This has been done in fig. 3 for X-rays and fig. 4 for neutrons.

It will be seen that parameters *b* and *c* are very closely correlated, though *c*, as might be expected, gives a lower estimate. For practical purposes therefore, when the magnitude of the correction for unfertilised eggs is unknown, *c* is preferable to *b*. Parameter *a* has special features. For X-rays it is closely equivalent to *b* up to values of unity, but above that it rises much more steeply with dose. This must be the result of the high contribution of multi-lethal eggs to ED at the highest mutation rates. Thus *a* is the best estimate at low doses, but the worst at high doses. With neutrons, *a* is *lower* than *b* at low

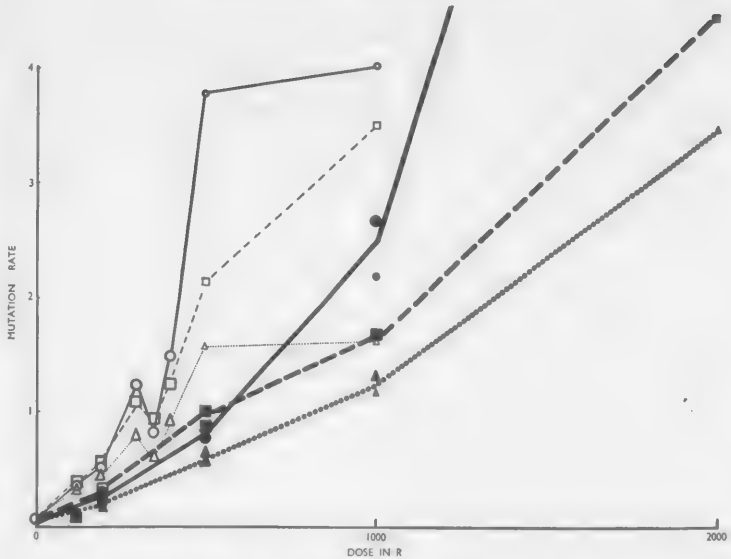


FIG. 3.—Dependence of various estimates of dominant lethal mutation rate on dose. Author's X-ray data. Solid symbols and thick lines: matings in the first two weeks after irradiation. Open symbols and thin lines: matings in third week. Small symbols: data in which (IMP−ED) >20. Circles and entire lines: ED/(IMP−ED). Squares and broken lines: $-ln\frac{(IMP-ED)}{CL}$. Triangles and dotted lines: $-ln(1-ED/IMP)$.

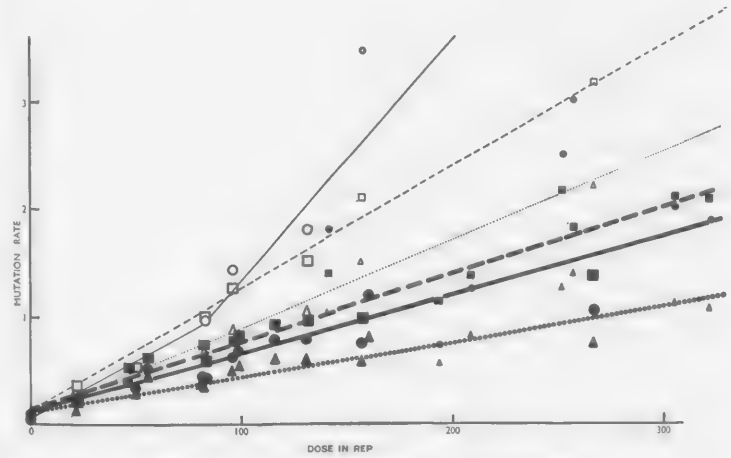


FIG. 4.—As in fig. 3, for Russell's *et al.* neutron data.

doses, owing to the greater incidence of multi-lethal eggs with neutrons, but again, when $b$ rises above unity, $a$ soars ahead.

In conclusion, when the proportion of unfertilised eggs is known, the gross estimate of mutation rate is best. When the proportion of unfertilised eggs is not known ED/(IMP−ED) is best for gross mutation rates of less than unity, but $-ln(1-\text{ED/IMP})$ is consistent over all dose ranges. ED/(IMP−ED) is also easier to calculate for biologists unaccustomed to natural logarithms.

## 5. DOSE DEPENDENCE

A striking difference between figs. 3 and 4 is that while for X-rays all the parameters show a concave dose-dependence curve, for neutrons all except ($a$) in the third week show a linear curve. This bears obvious resemblances to the well-known difference between X-rays and neutrons in respect of the dose dependence of rearrangements. It may be presumed that the difference has the same cause, namely, the induction of more than one break by a single neutron track. The departure from linearity for the dependence of dominant lethal mutations on X-ray dose is according to the predictions of Haldane and Lea (1947) in their theory of chromosome rearrangements.

## 6. SUMMARY

1. Three hypotheses are proposed to explain the time of death of dominant lethal embryos :

A. Embryos with one lethal produce deciduomata ; those with more than one are not implanted.

B. A constant fraction, about one half, of all dominant lethal embryos fail to survive to implantation.

C. (A compromise between A and B.) All embryos with one lethal, and about one-quarter of multi-lethal embryos produce deciduomata, the rest being unimplanted.

An additional assumption combined with all three hypotheses is that after X-rays the number of lethals per embryo is distributed in a Poisson series.

From the above it is possible to predict the relationship between deciduomata (ED) and the discrepancy between the numbers of corpora lutea and implants (PRE).

2. For my own X-ray data, the relation between ED and PRE excludes B and favours C over A.

3. For neutron data, after allowance has been made for the fact that the number of lethals per egg shows clustering instead of following a Poisson series, the relation between ED and PRE again favours A and C. It appears that about one-third of the lethal-inducing neutron tracks induces more than one lethal.

4. Three parameters for measuring the dominant lethal mutation rate are compared :

$$(a)\ \mathrm{ED}/(\mathrm{IMP}-\mathrm{ED}),$$

$$(b)\ -ln\left(\frac{1-\frac{\mathrm{PRE}+\mathrm{ED}}{\mathrm{CL}}}{0{\cdot}9}\right), \text{ and}$$

$$(c)\ -ln(1-\mathrm{ED}/\mathrm{IMP}).$$

5. The parameters are closely equivalent for actual data until $\mu$ rises above unity, when $(a)$ gives increasingly exaggerated estimates. It is concluded that $(a)$ is best for low rates but $(c)$ is best over the whole range unless the rate of unfertilised eggs is known and constant, when $(b)$ is best.

6. The dose dependence of dominant lethals shows strict proportionality for neutrons, but non-linearity for X-rays.

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# DIALLEL CROSS IN PLANT BREEDING

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A DIALLEL cross consists of all possible crosses between a number of varieties. Reciprocal crosses, and the selfed parents, may or may not be omitted. Such a set of crosses is obviously of interest to the plant breeder, but the information obtained may not be worth the trouble of making the cross. In this paper the utility of such crosses is considered, using data available in the literature together with some new figures from an $18 \times 18$ cross in tomatoes.

The statistical analysis of a diallel cross has been described by Yates (1947). It consists of fitting additive main effects for parents, and their interactions in the individual crosses. Such a main effect is sometimes called " general combining ability " or " additive genetical component " while an interaction may be referred to as " specific combining ability " or " non-additive genetical component ". The word interaction is used here in its purely statistical sense of a departure from additivity. It should not be confused with any form of genetical interaction between postulated " genes ". The interactions are part of the statistical description of the data, being the ups and downs which remain when the main effects have been taken out. The analysis is similar to that of factorial experiments, and merely assumes that the contributions of male and female parents are equally important. The exact mode of inheritance is not specified, and the analysis would be as effective for, say, blending inheritance as for Mendelian. There is no need for the parents to be inbred (or to have a uniform coefficient of inbreeding). It may be objected that a plant breeder is interested in, say, the top $2\frac{1}{2}$ per cent. of each progeny, and so wishes to estimate not the mean $\mu$ but $\mu + 2\sigma$ where $\sigma$ is the standard deviation. This, however, involves an adjustment in practice and not in principle. In many cases there will not be enough replication to give useful estimates of $\sigma$ for each particular cross and heterogeneity of variance will be undetectable. Main effects can, however, be fitted for log variance or standard deviation in the same way as for progeny means. This factorial method of analysis is to be judged by its success in describing the data.

## 1. THE " POLYGENE " ANALYSIS

A second method of analysis, based on ideas introduced in the classical paper of Fisher (1918) and developed by Fisher, Immer and Tedin (1932) and Mather (1949), has been put forward by Jinks and Hayman (1953), Jinks (1954) and Hayman (1954*b*). It involves the assumption, *inter alia*, that the yield (or whatever character is being

analysed) is controlled by a number of genes with additive effects, which may show dominance and interaction between loci. These genes are individually unrecognisable ; the analysis is concerned with their combined action as revealed by the various statistics. Now, no analysis is of any direct use to the practical breeder unless it causes him in some way to alter his breeding programme for the better. Before starting any experiment in any field of inquiry, the questions at issue and the relevance of the information to be gained should be clearly and explicitly stated. It is not at all clear how the " polygene " analysis can help the breeder in a specific problem since, even accepting the genetical assumptions and statistical methods employed, it is not known which plants contain which genes. Further knowledge of the genetics of continuous variation in general would, of course, be beneficial to all plant breeding.

The concepts of dominance, epistacy and so on are more complex in polygene analysis than in Mendelian genetics. For example, a study of the known genes in *Drosophila* (Bridges, 1944), maize (Weijer, 1952) and tomato (Rick and Butler, 1956) shows that non-lethal genes nearly always show complete dominance or (as in blood groups) complete independence, although Haldane (1954) points out that homozygotes and heterozygotes that are macroscopically indistinguishable may show quantitative biochemical differences. In polygene analysis the possibility of lethality is ignored and dominance is taken to mean any departure of the effect of A*a* from the average of the effects of AA and *aa*. As Mather (1949) has shown, a change of scale can convert " semi-dominance " into neutrality or recessiveness. This vagueness is the fault, not of the analysis itself, but of the difficulties arising from the nature of the data. But the consequences are, first, that the genetic labels such as dominance or complementarity are easily confused in the results obtained in practice (Hayman, 1954*b*) and, second, that alternative genetical systems characterised by, *e.g.* purely multiplicative gene effects could be analysed in the polygene way in terms of additive gene effects and their various types of interaction, *i.e.* dominance, epistacy, etc. The idea of additive independent gene effects—treating departures from this simple scheme as genic interaction—is employed solely because it leads to simplicity in the statistical analysis. Other hypotheses, *e.g.* multiplicative gene action, would be equally acceptable genetically. There is thus a danger of accepting this convenient working hypothesis as a paragon, and supposing that the genetics of continuous variation must necessarily be viewed in this way. One can say " the situation can be explained thus " but not " the situation evidently must be explained thus and not otherwise ". In qualitative genetics Mendelism is supreme and cases such as that of Irwin (1947), where a hybrid gives a product qualitatively different from that of either parent, appear to be rare. No such all-embracing scheme has yet been found in quantitative genetics. This is not to say, of course, that Mendelism is inapplicable

here ; there can be no doubt that it is applicable. But the mathematical applications of Mendelism so far tried have not been completely successful in producing a clear-cut picture. The general picture—*e.g.* the fraction of the observed variation which may reasonably be dubbed " genetical "—should turn out to be much the same whatever the genetical hypothesis, but the details will depend on the particular hypothesis used. In practical breeding work, therefore, it should not be supposed that the idea of additive gene action is an absolute way of looking at the situation, although it may well give as good a description as any. But even in an ideal situation in which it was certain that the system behaved—and would go on behaving—according to the additive hypothesis, the results of the polygene analysis of a diallel cross appear to have no relevance to the practical problems of the breeder. They would not help him decide what to do next. This does not, of course, affect the validity of such analysis as a tool in pure genetics ; but the analysis is also open to criticism on both genetical and statistical grounds. There is no reason why valid statistical methods should not be used, but the genetical objections are not easily disposed of.

The *genetical assumptions* are (Hayman, 1954*b*) :

(i) Diploid segregation
(ii) No difference between reciprocal crosses
(iii) Independent action of non-allelic genes

and in the diallel cross

(iv) No multiple allelism
(v) Homozygous parents
(vi) Genes independently distributed between the parents.

In his discussion Hayman shows that a failure of assumption (ii) can be dealt with and that assumption (iv) is unimportant until the $F_2$ but that failure of any of the others leads to trouble. He makes the recommendation—remarkable to a statistician—that should a statistical test (that in his notation the regression of Wr on Vr must be linear and of unit slope) fail, particular lines may be removed from the diallel cross until the remnant does satisfy this test. This procedure has been disputed by Kempthorne (1956) on the ground that, if the original set of parents is regarded as a random sample from some larger population, the reduced set of parents cannot be so regarded. This objection loses its force when a breeder is concerned only with the particular parents involved. This same point must be made about Griffing's (1956*a*) demonstration, following Fisher, that the inclusion of selfs as well as crosses causes bias. But the principle that, if a set of data contradict some limited hypothesis, the offending parts of the data may be removed and the analysis completed on the remainder, is debatable. It is usually thought better to reject the hypothesis as it stands. It might at least be expected that the significance levels employed when testing the remnant should be made

more stringent—following the usual probability laws—to allow for the selection that has been practised on the data. The gravity of this matter can be seen from the statement of Jinks (1954) that in one case an $8 \times 8$ cross was reduced to a $5 \times 5$. The results of such selective analysis cannot be supposed to have any wider, inductive application except to material that has been similarly selected.

The same objection applies to Hayman's alternative recommendation that a particular progeny may be disallowed, and missing-plot technique used to replace it, in order that the data may fit the hypothesis. The " missing plot " is merely a device to facilitate the analysis of an otherwise difficult (because incomplete) set of data and should not be used to replace egregious and unwelcome (but perfectly genuine) observations.

Assumption (i) of diploid segregation is usually perfectly justified. However, endosperm (which makes up the greater part of the yield of cereals) is triploid and in fact about one-third of the genes in a grain of maize are not derived from the plant on which it grew at all. Material of one genotype grows on a plant of another. The phenomenon of Xenia is well known for Mendelian characters; whether it applies to yield is unknown. This point is unimportant to Yates' (1947) method of analysis of first-order statistics but is relevant to Jinks and Hayman's (1953) analysis of second-order statistics of the yield of maize (or any other cereal). It is, however, a small point; at best the pollen " rain " will be uniform over the whole experiment, and there will merely be a small increase in the error component. Incidentally, it is not clear whether Kinman and Sprague (1945), from whom Jinks and Hayman derive their data, are reporting total ear weight or yield of shelled corn. This does not affect the present argument much since the corn contributes about $\frac{8}{9}$ of the weight of an ear of maize (Griffing and Lindstrom, 1954).

In biochemical gene action there must be some kind of negative feed-back to give stability of development. The only alternative is that each gene can make only a fixed amount of its product between divisions. Gene interaction cannot be avoided at the biochemical level and so assumption (iii) dealing with the phenotypic level, must be purely empirical. It can be tested and may be found to give an adequate description of the ultimate gene effects on size of plant. Assumption (v) is a consequence of inbreeding in the absence of inversions or other chromosomal devices for the maintenance of heterozygosity, when this is selectively advantageous to the plant.

Assumption (vi) is impossible except in trivial cases. For example, if in a $10 \times 10$ cross 3 parents are AA (and 7 *aa*), and 8 parents are BB (and 2 *bb*) then it is assumed that exactly 2·4 parents are AA BB. It is not clear to what extent the inevitable departures from this assumption are tolerable.

The mathematical consequences of failure of assumptions (ii), (iii), (iv) and (v) have been explored by Dickinson and Jinks (1956),

Kempthorne (1956) and Matzinger and Kempthorne (1956). These papers are interesting as theory. It is evident that, while it is quite possible to sort out the various kinds of interaction between the postulated " additive " genes, data that could genuinely do so would have to be much more extensive than any so far published. Once interaction between loci is established, the value of the concept of discrete additive genes is reduced. Kempthorne points out that assumption (vi) remains crucial, and he insists that the parents must be the result of unselected inbreeding from a random mating population. This ensures the approximate satisfaction of assumption (vi), although it is not strictly necessary to it. It is hard to think of any other justification for making such an assumption.

Now it might be asserted that such genetical assumptions are only made in order to obtain a simple working hypothesis (as is done in any statistical analysis of any type of data), and that the important point is whether the hypothesis fits the data. The way in which, in this case, the data are made to fit the hypothesis has been criticised above. By making a seventh assumption, of equality and absence of oppositions in the dominance effects of the various genes, it is possible to estimate the number of loci involved. Of the results of such estimation Jinks (1954) remarks " It would appear from these results that the basic assumptions on which this estimation is based are far from being realised in the present data and for that matter in any other data so far analysed by these methods." The failure of this seventh assumption alone would not invalidate the rest of the analysis, but it is not clear why it should necessarily be the seventh assumption that is blamed. This estimate of the number of genes is critical because, if there is any point in it at all, it must give a commonsense answer ; whereas the other parameters estimated may reasonably take almost any values.

## 2. STATISTICAL METHODS

The least squares estimation procedure proposed by Jinks (1956) is open to criticism on grounds of statistical efficiency. The matter has been discussed by Mather (1949) and by Nelder (1953). Fisher (1928) showed that the use of inefficient estimates produces misleading results and it is unusual to give such estimates unless it is certain that the loss of information is small. Efficient estimation (*e.g.* by maximum likelihood) is not impossible, although certainly more troublesome in this case than least squares. This objection does not apply to the analysis of $F_1$'s only since estimation is then by equation of observation to expectation.

A more serious matter is the regression of Wr on Vr. Vr is the estimated variance of the set (or " array ") of all crosses involving the $r^{th}$ parent (including that parent selfed). Wr is the covariance of the array with the corresponding parents. Obviously Wr and Vr do not fulfil the assumptions about independence and normality which are basic to regression analysis.

Hayman's (1954*b*) use of Wr+Vr to determine the order of dominance of the parents needs to be justified by the previous demonstration of overall heterogeneity in the values of Wr+Vr. Since Wr is statistically correlated with Vr a suitable though approximate procedure would be to test Vr for heterogeneity by Bartlett's (1937) test. (Although the Vr's are not statistically independent, because each cross contributes to two of them, this test still gives satisfactory results, at any rate for values of $k$ exceeding 6.)

Thus although "polygene" analysis should not be dismissed out of hand, the greatest possible circumspection should be used when considering its results. Theory is liable to outrun performance.

## 3. PRACTICAL RESULTS

I shall now take such data as are available and see what answers they give to a number of questions of interest to the breeder. These data are derived mainly from tomatoes and maize, both of which exhibit hybrid vigour. Heterosis in the inbreeding tomato is not of the same magnitude as in outbreeding maize, but it cannot be neglected by the breeder. Neither can it be ignored by the statistician, and so the $F_1$'s have been analysed separately from the parents throughout. The term $F_1$ is here used to distinguish the crosses from the parents: it does not necessarily imply that the parents were completely inbred. Since reciprocal crosses are not represented, the analysis is in essence that of the diagonal sums in section 2 of Yates (1947), with some extensions. The numerical results on which the conclusions are based are summarised in the Appendix.

### (i) *Is it helpful to describe the data in terms of main effects and interactions, and do such interactions occur?*

This question is answered by means of the analysis of variance. In a $k \times k$ diallel cross (omitting reciprocals) with each progeny replicated $r$ times, let $y_{12}$ = the sum (over $r$ plots) for the cross of parent 1 × parent 2, and $Y_1$ = the sum (over $(k-1)r$ plots) of all the $(k-1)$ $y$'s which involve parent 1. Then the analysis of variance of $F_1$'s only runs:

| | d.f. | S.S. |
|---|---|---|
| Parents (main effects) . . | $k-1$ | $\frac{\text{dev}^2 Y}{r(k-2)}$ |
| Interactions . . . . . | $\frac{1}{2}k(k-3)$ | by difference |
| Between $F_1$'s . . . | $\frac{1}{2}k(k-1)-1$ | $\frac{\text{dev}^2 y}{r}$ |

Here "dev²" means as usual "sum of squares about the mean". An error S.S. is derived as usual from replication of the parents and $F_1$'s. If the "main effects" mean square does not exceed the "interactions" M.S., the main effects are useless for predicting the yields of individual crosses and the concept of general combining ability is unhelpful. If the "interactions" M.S. exceeds the error M.S. then, however useful the main effects may be, they are not completely successful in prediction. In practice main effects provide an apt way of describing the behaviour of the crosses, but interactions do occur. This means that parents with relatively small main effects cannot be expected to contribute to a high-yielding cross. In the $18 \times 18$ cross, for example, many of the 153 interactions were "significant" but none was so large as to ruin completely the prediction from main effects for that particular cross. But it does not follow that the two parents with the largest main effects will necessarily combine to give the best cross; for they may show a negative interaction, or another cross may show a large positive interaction. It may turn out (as in fertiliser trials) that main effects are more constant from year to year than interactions, but there is as yet insufficient evidence on this point.

### (ii) *Do main effects give better prediction of $F_1$ yields than a regression on the mid-parent?*

The "mid-parent" is the mean of the yields of the two parents concerned. Let $P_1$ = the sum (over $r$ plots) for parent 1. The calculation of the regression of $y_{12}$ on $(P_1+P_2)$ can be shortened by using the algebraic identities $\text{dev}^2(P_1+P_2) = (k-2)\text{dev}^2(P_1)$ and $Sy_{12}(P_1+P_2) = \Sigma Y_1 \,.\, P_1 - \frac{(\Sigma Y)(\Sigma P)}{k}$. This sum of products may also be used in estimating the correlation between main effects and parental yields, but I shall not do so here because the correlation as such is less pertinent to the breeder than questions (ii) and (iii) under discussion. The variance of the $\frac{1}{2}k(k-1)$ $y$'s is then split up in the usual way into 1 d.f. for regression and $\frac{1}{2}k(k-1)-2$ d.f. remainder. The "regression" component is in fact part of the "main effects" S.S. in (i), being orthogonal to the "interactions" S.S. If the "remainder" M.S. turns out to be consistently smaller than the "interaction" M.S., then regression gives better prediction than the main effects. In practice it does not.

It can be argued that the main effects ($t$) have an error variance $\frac{\sigma^2}{(k-2)r}$ whereas the P's, being the total of only $r$ plots, are less accurately estimated. This purely statistical difference can be corrected for (approximately) by subtracting $\frac{(k-1)(k-3)rs^2}{k-2}$ from $\text{dev}^2P$, where $s^2$ is the error M.S. When reciprocals are included in the experiment, the main effects have error variance $\frac{\sigma^2}{2(k-2)r}$ and the

correction to dev²P is $\frac{(k-1)(2k-5)}{2(k-2)}\,rs^2$. A regression analysis carried out with this corrected value of dev²P will allow for this statistical difficulty, although it cannot be expected to follow the mathematical theory of regression exactly. This does not matter much here, where the problem is not to estimate a regression coefficient but to see whether the superiority of the main effects for prediction is merely due to their superior accuracy of estimation. In practice it is not.

(iii) *Within the limits of experimental error, do the main effects correspond to the parental yields, or are some parents more potent when crossed than would be expected from their own yields ?*

This question overlaps question (ii) and is not of great practical value in individual experiments since a breeder is interested in the magnitude of the main effect as such rather than its relation to the parent yield. But it is of interest when considering the general utility of the diallel cross. Since $y_{12} = m+t_1+t_2+t_{12}$ (where $m$ is the general mean, $t_1$, $t_2$ are main effects and $t_{12}$ the interaction), it follows that the difference between $y_{12}$ and its mid-parent is

$$m+\left(t_1-\frac{p_1}{2}\right)+\left(t_2-\frac{p_2}{2}\right)+t_{12},$$

where $p$ is the parental mean. The question is therefore whether the values of $(t-p/2)$ are heterogeneous. To answer this, the value of $2\mathrm{Y}-(k-2)\mathrm{P}$ is found for each parent. Then $\frac{\mathrm{dev}^2[2\mathrm{Y}-(k-2)\mathrm{P}]}{(k^2-4)r}$ is a " heterogeneity of potence " S.S. with $(k-1)$ d.f. Its mean square can be compared with the error M.S. When reciprocals are included in the experiment, this expression becomes $\frac{\mathrm{dev}^2[\mathrm{U}-(k-2)\mathrm{P}]}{k(k-2)r}$ (Hayman 1954*a*) where U corresponds to Y but is now the sum of $2(k-1)r$ plot-observations. In practice such heterogeneity of potence does occur, and is of the same order of magnitude as the interactions (but not the main effects). This heterogeneity is unpredictable in that the regression of main effect on parental mean does not, on the average, differ from $\frac{1}{2}$. Now if the answers to questions (ii) and (iii) had been negative, there would be no point in bothering about main effects at all since the yields of the parents themselves would do as well. But the present results mean that quite apart from the matter of interaction, the diallel cross does contribute information that cannot be obtained from the parents as such ; but this additional information is not equal in importance to the parental yields. To paraphrase a famous quotation, like begets like ; but some are more like than others.

### (iv) *The relation between $F_1$ and $F_2$*

In one case (section 1 of the Appendix) figures are available for $F_1$ and $F_2$. It is of course uncertain whether the relationship between the data of the two generations in this particular case will be found to be a general one. The relation between $F_1$ and $F_2$ has been ably discussed by Hayman (1957). Although that paper is couched in polygene terms, it is primarily concerned with first-order statistics (means) rather than with second-order (variances and covariances). This means not only that the methods employed are more reliable from a purely statistical point of view, but that they do not in fact require all the assumptions usually involved in polygene analysis. They would be adopted by those who favour a more cautious and pragmatic approach such as is advocated here. The technical considerations relevant to first- and second-order statistics have been discussed by Nelder (1953). Further $F_2$ material is reported by Jinks (1956), but I have not been able to analyse his (unpublished) original data in this alternative fashion.

It is not surprising to observe a reduction in the average size of the interactions in the $F_2$ (as measured by the mean square). The correlation between corresponding $F_1$ and $F_2$ interactions is small but positive ($r = 0{\cdot}255$, 34 d.f.). The regression of $F_2$ main effects on parental means and $F_1$ main effects gives

$$F_2 \text{ main effect} \simeq 0{\cdot}41(\tfrac{1}{2}p) + 0{\cdot}53\,t$$

and does not differ significantly from the expected $\frac{1}{2}(\frac{1}{2}p+t)$. Such expected main effects are almost as good as the estimated $F_2$ main effects for predicting the relative yields for $F_2$ progenies :

| | d.f. | M.S. |
|---|---|---|
| (*a*) Regression on $\frac{1}{2}(\frac{1}{2}p+t)$ expected main effects | 1 | 2790·1 |
| Remainder | 43 | 31·2 |
| (*b*) Regression on individual $\frac{1}{2}$(mid-parent+$F_1$) | 1 | 2455·9 |
| Remainder | 43 | 39·0 |
| (*c*) $F_2$ estimated main effects | 9 | 363·2 |
| Interactions | 35 | 24·7 |

The complete diallel cross is, in fact, more likely to be worthwhile in the investigation of such relationships between generations in continuous variables than in a breeding programme as such. If the number of parents involved is large enough to interest the breeder, the number of possible crosses reaches a formidable total and some device for reducing the amount of work involved may become necessary.

## 4. FRACTIONAL REPLICATION

It appeared above that in practice the main effect, or general combining ability, cannot be predicted exactly from the parental mean. Furthermore, the parents themselves give no information

about the possibilities of heterosis. The breeder may therefore wish to estimate the main effects of a set of $k$ varieties without making all the $\frac{k(k-1)}{2}$ possible crosses. An obvious course is to use three or four control varieties and cross them with all the experimental ones. This is wasteful if the control varieties are of no direct interest to the breeder, since each cross contributes information about one main effect only (instead of two) and the interactions obtained are valueless. In the more likely case when the "control" varieties are in fact experimental material, the "control" main effects are very well estimated and the others poorly. It is possible to make a more balanced experiment at the price of a more complicated statistical analysis.

Complete balance is unobtainable; that is to say, unless every cross is grown, the differences between the various main effects will not all be estimated with equal accuracy. But the discrepancies will not be very serious if the number of crosses to which each parent contributes is constant. It is not difficult to satisfy this condition. The problem is connected with a particular type of Latin square: or alternatively it may be considered as that of arranging $k$ varieties in incomplete blocks of two plots each. If $k$ is divisible by 4, the simplest solution is given by a $k \times k$ Latin square symmetrical about both diagonals; *e.g.* for $k = 8$, the upper half of a square such as

| | | | | | | | | or | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| h | a | b | c | d | e | f | g | | h | a | b | c | d | e | f | g |
| a | h | g | b | e | d | c | f | | a | h | c | b | e | d | g | f |
| b | g | h | a | f | c | d | e | | b | c | h | f | a | g | d | e |
| c | b | a | h | g | f | e | d | | c | b | f | h | g | a | e | d |
| d | e | f | g | h | a | b | c | | d | e | a | g | h | f | b | c |
| e | d | c | f | a | h | g | b | | e | d | g | a | f | h | c | b |
| f | c | d | e | b | g | h | a | | f | g | d | e | b | c | h | a |
| g | f | e | d | c | b | a | h | | g | f | e | d | c | b | a | h |

is superimposed on the possible crosses

| | | | | | | |
|---|---|---|---|---|---|---|
| 12 | 13 | 14 | 15 | 16 | 17 | 18 |
| | 23 | 24 | 25 | 26 | 27 | 28 |
| | | 34 | 35 | 36 | 37 | 38 |

etc.,

and each parent occurs once in the four crosses picked out by any letter of the Latin square. If $k$ is even, but not divisible by 4, similar designs without the same degree of symmetry are easily found; for $k = 6$ the only three complete solutions are

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| f | a | b | c | d | e | | f | a | b | c | d | e |
| a | f | e | b | c | d | | a | f | c | d | e | b |
| b | e | f | d | a | c | | b | c | f | e | a | d |
| c | b | d | f | e | a | | c | d | e | f | b | a |
| d | c | a | e | f | b | | d | e | a | b | f | c |
| e | d | c | a | b | f | | e | b | d | a | c | f |

and the mirror image of the second square about its second diagonal. Incomplete solutions are also possible, *e.g.*

| | | | | |
|---|---|---|---|---|
| a | d | b | d | c |
| | c | d | b | d |
| | | a | d | b |
| | | | c | d |

where each parent occurs once in a, b, c but twice in the set d which cannot be split up into two single sets. If $k$ is odd, each parent must be represented an even number of times. Different sets of crosses in which each parent occurs twice may be found for any value of $k$ by writing down the parents in order and taking consecutive pairs ; thus

1 2 3 4 5 gives 12, 23, 34, 45, 51

and

3 1 4 2 5 gives 31, 14, 42, 25, 53

The statistical analysis of such an incomplete diallel cross involves the solution of a fairly tractable set of non-orthogonal least squares equations. Thus for $k = 6$ the set of crosses 12, 13, 14, 24, 25, 35, 36, 46, 56 gives least squares and reciprocal matrices

$$\begin{pmatrix} 3 & 1 & 1 & 1 & 0 & 0 \\ 1 & 3 & 0 & 1 & 1 & 0 \\ 1 & 0 & 3 & 0 & 1 & 1 \\ 1 & 1 & 0 & 3 & 0 & 1 \\ 0 & 1 & 1 & 0 & 3 & 1 \\ 0 & 0 & 1 & 1 & 1 & 3 \end{pmatrix} \text{ and } \frac{1}{72} \begin{pmatrix} 37 & -11 & -17 & -11 & 7 & 7 \\ -11 & 37 & 7 & -11 & -17 & 7 \\ -17 & 7 & 37 & 7 & -11 & -11 \\ -11 & -11 & 7 & 37 & 7 & -17 \\ 7 & -17 & -11 & 7 & 37 & -11 \\ 7 & 7 & -11 & -17 & -11 & 37 \end{pmatrix}$$

This idea of fractional replication is not so useful as it may appear at first sight. The omission of some crosses will result in an alteration in the estimated main effects, due to the interactions of the omitted crosses. This will not, however, invalidate the fractional replicate since the interactions are less important than the main effects. (If this were not the case, the concept of main effects and interactions would have to be abandoned.) The fractional replicate merely offers a rational solution to the problem of a breeder who wishes to grow some exploratory crosses without making all possible crosses. The main effects can then be used to give expectations for the missing crosses, but clearly it would be better to grow all crosses if possible.

## 5. CONCLUSIONS AND SUMMARY

The diallel cross offers a means of rationalising some aspects of plant breeding while keeping the amount of work down to a manageable level. Its utility to the breeder can be exaggerated. The polygene analysis of a diallel cross suffers from several theoretical defects, but in any case its results do not appear to be directly relevant to practical breeding work. The performance of the parental varieties themselves

gives valuable prediction of the relative behaviour of the crosses, but the diallel cross does give further information. Whether it might be used for assessing long-term potentialities of raw material at the beginning of a long breeding programme is unknown.

A breeder faced with a mass of material would do well to make an ordinary variety trial to discover the best 15 or 20 lines. He might then employ these as parents in a fractional diallel cross with each parent represented in perhaps 8 crosses. Alternatively, he might eliminate more parents on the basis of their performance as varieties and then make, say, a complete $10 \times 10$ cross. In either case, the results would permit the selection of a few parents for intensive work. This is, of course, a naïve idealisation of a breeder's position, but shows that the diallel cross can be used to advantage in the later stages of selection. No statistics can replace the breeder's intimate knowledge of his crop : but they can help.

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## APPENDIX

Those parts of the numerical analyses relevant to the discussion are reproduced here in a condensed form. They include

(i) The analysis of variance of $F_1$'s by main effects, interactions and error (where available).

(ii) The analysis of variance of $F_1$'s by regression on the mid-parents (*a*) as they stand and (*b*) correcting for the inferior accuracy of parental yields compared with the main effects.

(iii) The mean square for heterogeneity of potence.

1. Kinman and Sprague (1945). Yield of inbred parents, $F_1$ and $F_2$ in maize. $k = 10$, $r = 1$.

| | d.f. | M.S. for $F_1$ | M.S. for $F_2$ |
|---|---|---|---|
| (i) Main effects | 9 | 424·2 | 363·2 |
| Interactions | 35 | 118·0 | 24·7 |
| (ii) (*a*) Regression | 1 | 2296·0 | 2372·6 |
| Remainder | 43 | 131·4 | 41·0 |
| (iii) Heterogeneity | 9 | 59·4 | 35·7 |

2. Griffing and Lindstrom (1954). Ear weight and shelled corn weight in maize. $k = 9$, $r = 6$. Part of this analysis has been presented by Griffing (1956*b*.) The parents were randomised separately from the $F_1$'s, but this does not invalidate the present analysis.

| | d.f. | M.S. for ear wt. | M.S. for corn wt. |
|---|---|---|---|
| (i) Main effects | 8 | 90·98 | 1948·7 |
| Interactions | 27 | 6·99 | 260·5 |
| Error | 175 | 3·02 | 116·1 |
| (ii) (*a*) Regression | 1 | 350·55 | 7116·4 |
| Remainder | 34 | 16·65 | 456·1 |
| (*b*) Regression | 1 | 387·01 | 7810·3 |
| Remainder | 34 | 15·57 | 435·7 |
| (iii) Heterogeneity | 8 | 17·19 | 461·0 |

3. Allard (1956). Seed-size of lima beans. $k = 9$, $r = 5$. The experimental layout was not randomised, so that the estimate of error is not strictly valid and progeny means may be biased.

| | d.f. | M.S. |
|---|---|---|
| (i) Main effects | 8 | 331·8 |
| Interactions | 27 | 51·6 |
| Error | 144 | 10·2 |
| (ii) (*a*) Regression | 1 | 1986·2 |
| Remainder | 34 | 60·6 |
| (*b*) Regression | 1 | 2144·3 |
| Remainder | 34 | 56·0 |
| (iii) Heterogeneity | 8 | 31·0 |

4. Hayman (1954). Flowering date of *Nicotiana rustica*. $k = 8$, $r = 2$. Reciprocals were included and the following analysis is of reciprocal sums (and differences) as described by Yates (1947).

| | d.f. | M.S. |
|---|---|---|
| (i) *Reciprocal sums* | | |
| Main effects | 7 | 175·05 |
| Interactions | 20 | 21·20 |
| *Reciprocal differences* | | |
| Main effects | 7 | 53·43 |
| Interactions | 21 | 11·66 |
| Error | 63 | 3·37 |
| (ii) (*a*) Regression | 1 | 997·89 |
| Remainder | 26 | 25·06 |
| (*b*) Regression | 1 | 1086·11 |
| Remainder | 26 | 21·66 |
| (iii) Heterogeneity | 7 | 8·52 |

5. Jinks (1954). Plant height of *Nicotiana rustica*. $k = 8$, $r = 2$. Parents and $F_1$ reported for two successive years. Means of reciprocals are given, and the error is estimated from reciprocal differences, not replication. This estimate of error appears to be on a per-plot basis and is quoted here as such.

| | d.f. | M.S. 1st year | M.S. 2nd year |
|---|---|---|---|
| (i) Main effects | 7 | 1014·67 | 902·27 |
| Interactions | 20 | 59·61 | 43·80 |
| Error | 56 | 2·49 | 7·71 |
| (ii) (*a*) Regression | 1 | 5661·64 | 4606·11 |
| Remainder | 26 | 101·28 | 99·46 |
| (*b*) Regression | 1 | 5820·38 | 4975·64 |
| Remainder | 26 | 91·79 | 85·24 |
| (iii) Heterogeneity | 7 | 91·12 | 80·23 |

6. Currence, Larson and Virta (1944). Yield of tomatoes. $k = 6$, $r = 2$.

| | d.f. | M.S. |
|---|---|---|
| (i) Main effects | 5 | 6·067 |
| Interactions | 9 | 1·775 |
| Error | 20 | 0·484 |
| (ii) (*a*) Regresssion | 1 | 23·480 |
| Remainder | 13 | 1·756 |
| (*b*) Regression | 1 | 28·099 |
| Remainder | 13 | 1·401 |
| (iii) Heterogeneity | 5 | 0·917 |

7. Powers (1945). Yield of tomatoes. $k = 10$, $r = 10$. Data for seven characters of less importance to the breeder are also given.

| | d.f. | M.S. |
|---|---|---|
| (i) Main effects | 9 | 1,456,000 |
| Interactions | 35 | 35,200 |
| Error | 486 | 18,200 |
| (ii) (*a*) Regression | 1 | 11,478,900 |
| Remainder | 43 | 66,500 |
| (*b*) Regression | 1 | 12,497,000 |
| Remainder | 43 | 42,800 |
| (iii) Heterogeneity | 9 | 144,700 |

8. English commercial varieties of tomatoes. $k = 18$, $r = 6$.

| | d.f. | M.S. for flowering date | M.S. for 4 weeks' yield |
|---|---|---|---|
| (i) Main effects | 17 | 82·58 | 900·7 |
| Interactions | 135 | 17·76 | 84·0 |
| Error | 528 | 3·33 | 40·2 |
| (ii) (*a*) Regression | 1 | 252·81 | 8744·4 |
| Remainder | 161 | 22·04 | 118·6 |
| (*b*) Regression | 1 | 296·99 | 10,448·3 |
| Remainder | 161 | 21·76 | 107·3 |
| (iii) Heterogeneity | 17 | 19·17 | 89·2 |

9. Statistics (variance components) derived from numerous analyses of variance (i) are quoted by Sprague and Tatum (1942) and Rojas and Sprague (1952) for maize and by Horner and Lana (1957) for tomatoes. These figures show the same general features as those quoted here.

# MALE PARTHENOGENESIS IN BARLEY

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LITTLE is known of the role of the cytoplasm in heredity and development. Relevant information has been obtained to a large extent from studies of lower organisms, there being fewer opportunities in higher plants for assessing its importance or for evaluating the interrelations of nucleus and cytoplasm. A greater understanding of the role of cytoplasmic constituents and their organisation, would be of considerable value in elucidating further the nature of gene action and the process of differentiation.

An opportunity of recognising the role of the cytoplasm arose from a study of the anomalous progeny of an interspecific cross of two members of the genus *Hordeum*. In the course of a series of attempts to overcome the high degree of interspecific incompatibility existing within the genus, (Davies, 1956), a naturally occurring autotetraploid form of *Hordeum bulbosum* ($2n = 28$) was crossed with a synthetic autotetraploid form of *Hordeum vulgare* ($2n = 28$). From the one hundred and fifty-five flowers of the former species pollinated, seven viable seed were obtained, and three plants survived to maturity.

## 1. MORPHOLOGY OF PARENTS AND PROGENY

*H. bulbosum* is a tall, prolifically tillering form, which, in its early stages of growth, has a prostrate spreading habit. *H. vulgare* on the other hand has fewer tillers, thicker stems, is shorter and more upright (plate, fig. 1). The three progeny are shown in plate, fig. 2 and will be referred to in the text henceforth as Plants 1, 2 and 3 respectively, commencing from the left-hand side of the figure.

Plants 1 and 2 were similar, and both very closely resembled diploid *H. vulgare*. They differed in only one respect—many of the stems branched at the first node below the ear (plate, fig. 3). Plant 3 was also somewhat similar to diploid *H. vulgare* in some, though not all, respects. It flowered very much later, and tillered more profusely, than the latter. It had the characteristic initial prostrate habit, and the dark green leaf colour of *H. bulbosum*. The conformation of some of the ears was also anomalous, in that they had the long awned glumes, and awned lateral florets of *H. bulbosum*. These anomalous ears also invariably exhibited other upsets. Some florets were underdeveloped, others had no ovaries and others had fewer than the normal number of stamens.

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The main features of parents and progeny can be summarised thus :—

| Character | Female parent *H. bulbosum* | Male parent *H. vulgare* | Progeny | | |
|---|---|---|---|---|---|
| | | | Plant 1 | Plant 2 | Plant 3 |
| *Height* | Tall | Short | Short | Short | Short |
| *Early habit* | Prostrate | Upright | Upright | Upright | Prostrate |
| *Stem thickness* | Thin | Thick | Thick | Thick | Thick |
| *Tiller number* | High | Low | Low | Low | High |
| *Pubescence* | Pubescent | Glabrous | Glabrous | Glabrous | Glabrous |
| *Auricle colour* | White | Red | Red | Red | Red |
| *Mildew resistance* | Resistant | Susceptible | Susceptible | Susceptible | Susceptible |
| *Glume awn length* | Long | Short | Short | Short | Long in some ears |
| *Lateral florets* | Awned | Blunt | Blunt | Blunt | Awned in some ears |
| *Abnormal features* | None | None | Branching stems | Branching stems | Upset in floral development of some ears |

## 2. CYTOLOGICAL STUDIES

The two parental forms were autotetraploids ($2n = 28$). Studies of mitotic configurations in the root tips, and meiotic configurations in the pollen mother cells revealed that the three plants obtained on crossing the two autotetraploids were diploids ($2n = 14$).

Chromosome pairing was complete in all the progeny (table 1). The low chiasma frequency of Plant 3 may have been due to environmental effects, or it may have been another symptom of the developmental upsets that occurred in this plant. Meiosis occurred at the same stage of floral development in the three progeny as in *H. vulgare*.

*Result of hybridisations.* Seed setting was complete in all three progeny, on selfing, and on crossing reciprocally with diploid *H. vulgare*. The progeny produced on selfing and crossing were completely normal in all respects, resembling *H. vulgare* and showing none of the anomalies of the original aberrant forms. Their chiasma frequencies at meiosis were as high as in normal diploid *H. vulgare*.

Backcrossing Plants 1, 2 and 3 on to the tetraploid *H. bulbosum* did not prove any more successful than crossing diploid *H. vulgare* with tetraploid *H. bulbosum*. 60·54 per cent. of the florets set seed but all embryos died within 28 days of fertilisation, as in the normal cross. No differences in compatibility with *H. bulbosum* existed between the three progeny.

## 3. DISCUSSION

Evidence obtained from the morphological and cytological studies, and the crossing programme indicates that the three plants investigated had arisen as the result of a parthenogenetic development of a diploid

nucleus from the pollen grain of tetraploid *H. vulgare*. On attempting to cross the latter with *H. bulbosum*, union of the sperm nucleus with the egg was precluded yet presumably the other generative nucleus fused with a nucleus in the embryo sac, as the seeds were well filled, indicating normal endosperm development.

A few examples of male parthenogenesis have been previously recorded—haploid parthenogenesis in *Nicotiana Landsdorffii* (Kostoff, 1929) and *Nicotiana tabacum* (Clausen and Lammerts, 1929), and diploid parthenogenesis in *Fragaria virginiana* (Ichyima, 1930) and in *Euchlaena mexicana* (Collins and Kempton, 1916).

In examples of male parthenogenesis, the nuclear material of the progeny is derived from the male parent, and the cytoplasm, it is

TABLE 1

| Parent | Chromosome no. (2n) | Mean metaphase configuration per cell | | | | Mean no. of chiasmata per cell | No. of cells analysed | Per cent. pollen fertility |
|---|---|---|---|---|---|---|---|---|
| | | Quadrivalents | Trivalents | Bivalents | Univalents | | | |
| *H. bulbosum* . | 28 | 4·66 | ... | 4·67 | 0·05 | 26·375 | 120 | 95·6 |
| *H. vulgare* . | 28 | 3·087 | 0·2174 | 7·13 | 0·739 | 23·348 | 23 | 92·1 |
| Plant 1 . | 14 | ... | ... | 7 | ... | 14·166 | 30 | 96·1 |
| Plant 2 . | 14 | ... | ... | 7 | ... | 14·203 | 30 | 94·7 |
| Plant 3 . | 14 | ... | ... | 7 | ... | 12·6 | 30 | 97·4 |
| *H. vulgare* . | 14 | ... | ... | 7 | ... | 14·37 | 24 | 94·3 |
| *H. bulbosum* . | 14 | ... | ... | 7 | ... | 12·738 | 80 | 97·2 |

assumed, is derived, at least initially, from the female. Hall's work (1954) on increasing the compatibility of species by changing the environment of the maternal plant in the embryo stage, might have led one to expect an increased compatibility of these male parthenogenetic progeny with *H. bulbosum*, but this result was not obtained.

The conclusion of male parthenogenesis must however be reconciled with the abnormal features observed in Plants 1, 2 and 3. It is unlikely that they could be attributed to nuclear gene mutation—one would have to assume a very high mutation rate, and the selective elimination of all mutant gametes or zygotes in the second generation, as all the progeny of the aberrant plants were normal. The possibility of chromosome breakage occurring in the embryo sac, followed by a transference of some *H. bulbosum* chromatin to the *H. vulgare* nucleus cannot be ignored, but a similar selective elimination would have to occur here also. Again this latter explanation becomes less plausible when one considers that some of the unusual features of the anomalous plants were not characteristic of the female parent, *H. bulbosum*.

The most plausible hypothesis regarding the origin of the anomalies or developmental upsets is that they were due to a disharmony of *H. vulgare* nuclear products and the *H. bulbosum* cytoplasmic environment. That the abnormalities were not unduly pronounced, indicates

that the two species had a very similar biochemical background. The normal development of the artificially cultured triploid hybrid of diploid *H. vulgare* and tetraploid *H. bulbosum* substantiates this conclusion (Davies, *loc. cit.*).

If the upsets observed could be attributed to nucleo-cytoplasmic disharmonies, then this conclusion must be reconciled with the existence of differences between the three parthenogenetic progeny, and between different parts of a given plant. All the gametes had a similar nuclear constitution and all three plants commenced development in the same ear of *H. bulbosum* and hence had a common cytoplasmic environment.

Differences between and within plants could be attributed to qualitative or quantitative differences in cytoplasmic factors. If these factors are assumed to have been particulate and capable of segregation, the occurrence of aberrations in one part and not another is easily accounted for. Alternatively the differences could have been due to changes in the environment at the time of differentiation, inducing a given state of stable equilibrium in one part, and another state in a second—this latter alternative, however, does not seem too plausible.

If we assume qualitative differences in these particulate cytoplasmic factors, or plasmagenes (Darlington and Mather, 1949) we imply the existence of numerous kinds, some of which exerted their influence during the development of ears, others of awns and others of tillers, etc. An alternative explanation could be that of quantitative rather than qualitative differences whereby cells had varying numbers of plasmagenes—an excess of "foreign" plasmagenes from *H. bulbosum* causing an anomaly in the differentiation and development of tissue having *H. vulgare* nuclei.

Quantitative differences in plasmagenes per cell could arise in any of the following ways :

(*a*) The irregular segregation of "foreign" plasmagenes at cell division. Such an aberrant segregation is thought to be the reason for the production of a respiratory mutant in yeast (Slonimski and Ephrussi, 1949).

(*b*) A differential survival of "foreign" plasmagenes between cells.

(*c*) If these cytoplasmic particles could reproduce even in the absence of the nucleus with which they were previously associated, then the non-uniformity within plants could arise from the lack of synchronisation of cell and particle division. This postulate is one that has been developed to account for certain observations of the kappa factor in *Paramecium* and the carbon-dioxide sensitive factor in *Drosophila*.

If this last alternative is correct then it is difficult to understand why the "foreign" plasmagenes were not perpetuated in the next generation of plants. They must either have (*a*) failed to persist through the sporogenous meiotic divisions, or (*b*) been suppressed prior to gamete formation.

*Plate*

FIG. 1.—Autotetraploid *Hordeum vulgare* on the left, autotetraploid *Hordeum bulbosum* on the right. × 1/10.

FIG. 2.—The three diploid male parthenogenetic progeny produced on attempting to cross the two plants shown in fig. 1. × 1/10.

FIG. 3.—A branching stem typical of two of the male parthenogenetic progeny, and a normal stem for comparison. × 1/6.

FIG. 4.—Spikes and florets of parental forms and of one of the male parthenogenetic progeny (plant 3). × 1/2.

A. *Hordeum vulgare*, with blunt lateral florets and short awned glumes.
B. *H. bulbosum*, with awned lateral florets, and long awned glumes.
C. Plant 3, with awned lateral florets, and long awned glumes.

1

2

3

4

These postulates have assumed the existence of " foreign " plasmagenes, in other words each nucleus is associated with a particular form of plasmagene. Sewall Wright (1941) has suggested how this could arise—inactive plasmagenes exist in the cytoplasm and these become activated by combination with prosthetic groups of nuclear origin. Alternatively a nucleus may act as a selective agent on a population of plasmagenes—favouring the reproduction of one type at the expense of another.

The alternative explanations offered, regarding the cause of the anomalies observed, and the differences between and within plants can be no more than hypotheses—the problem is not open to an experimental elucidation. Observations such as these, however, serve to indicate how the cytoplasm can exert an influence in its own right, but opportunities of recognising this influence occur very rarely. Even in the previously recorded instances of male parthenogenesis it has not been observed. Associations of the nucleus of one species with the cytoplasm of another, have occurred previously—the merogon of *Triton palmatus* and *Triton cristatus* being one example. This develops under the control of the maternal cytoplasm for a period of time, but succumbs eventually when the nucleus begins to exert its effect, due to an incompatibility of nuclear products and cytoplasmic constituents. That this did not occur in the present instance could have been due to a fundamental biochemical similarity of the two species concerned, or an early suppression of the incompatible cytoplasmic factors. Little is known of the specificity of any given population of such plasmagenes, and of processes of competition, adaptation, segregation, selection and dominance which may occur between them. As more information accumulates from studies of cytoplasmic inheritance in lower and higher organisms, of the processes of transplantation, grafting, hybridisation and infection, and ultimately the process of differentiation, a further evaluation of the phenomena observed in the present and similar instances will be possible.

## 4. SUMMARY

1. Male parthenogenetic progeny, produced on pollinating autotetraploid *Hordeum bulbosum* with autotetraploid *Hordeum vulgare*, were studied morphologically and cytologically.

2. Anomalous features within these plants could not be attributed to nuclear or environmental effects, but were probably due to a disharmony of nuclear and cytoplasmic constituents.

3. An explanation of the differences between and within the plants was sought in terms of quantitative variations of cytoplasmic factors.

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# ANIMAL BREEDING AND THE ESTIMATION OF GENETIC VALUE

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## 1. INTRODUCTION

In the field of animal breeding, the determination of optimum selection methods requires that the genetic value ($Y$) of an individual ($A$) be estimable from such data as—$A$'s phenotypic value ($y$), the mean phenotypic values of $A$'s $n$ full sibs ($\bar{F}$) ; $A$'s $m\bar{n}$ half sibs ($\bar{H}$), comprising $m$ families of full sibs, the $i^{\text{th}}$ with $n_i$ members, $\bar{n} = (\sum n_i)/m$ ; $A$'s $p\bar{q}$ progeny ($\bar{P}$), comprising $p$ families of full sibs, the $i^{\text{th}}$ with $q_i$ members, $\bar{q} = (\sum q_i)/p$ ; and such combinations of these as $\bar{F}' = (y+n\bar{F})/(1+n)$, $\bar{S} = (n\bar{F}+m\bar{n}\bar{H})/(n+m\bar{n})$ and $\bar{S}' = (y+n\bar{F}+m\bar{n}\bar{H})/(1+n+m\bar{n})$. This estimate is obtainable on the assumption of a suitable genetic model and an application of the statistical technique of multiple regression to $Y$ and the subset of the means $y$, $\bar{F}$, $\bar{H}$, $\bar{P}$, $\bar{F}'$, $\bar{S}$, $\bar{S}'$ on which it is to be based.

As is evident from the work of Lush (1945), Lerner (1950) and Osborne (1957), the genetic model almost invariably adopted is one in which the phenotypic value of an individual is considered as due to independent additive factors with superimposed non-interacting random and non-random environmental effects, and, although more sophisticated formulations have been developed, this simple model appears to be adequate in many situations and is, in practice, the basis of most agricultural advice as to optimum breeding plans.

However, there has been no systematic treatment of the various possible cases of interest and, although most of the simple regression formulae have been presented at one time or another, those of multiple regression have been generally neglected, as witness Lerner *et al.* (1947) requiring the partial regression coefficients of $Y$ on $y$, $\bar{F}$ and $\bar{P}$ and of $Y$ on $\bar{F}$ and $\bar{P}$ and applying instead the simple regression coefficients. In fact, the only case of multiple regression fully discussed has been that of the estimation of $Y$ from $y$ and $\bar{F}$, Lush (1947), and although this has been extended by Osborne (1957) to the estimation of $Y$ from $y$, $\bar{F}$ and $\bar{H}$ the discussion is subject to the restrictions that half sib families are all of equal size (i.e. $n_i = \bar{n}$ for all $i$) and non-random environmental effects are absent.

In the following sections, therefore, the various cases of estimation are considered systematically and explicit formulæ for the regression coefficients and the relative selective advance are derived, arbitrary

numbers of relatives and non-random environmental effects being allowed. The necessary modifications when all individuals are inbred are also indicated.

## 2. GENETIC MODEL

Each individual involved in the estimation is considered to be a member of a hypothetical population in which phenotypic value, $y$, is given by—

$$y = M+g_1+g'_1+g_2+g'_2+\ldots+g_k+g'_k+u+c+e,$$

the joint variations of these individuals being subject only to restrictions consequent on genetic relationship and common environment. $M$ is a constant and the other symbols random variables, $g_i$ and $g'_i$ describing the effects of the two genes representing the $i^{\text{th}}$ factor, $u$ and $c$ non-random and $e$ a random environmental effect. The variables $g_i$ and $g'_i$ may assume values $a_i$ or $b_i$ with probabilities $p_i$ and $q_i$, respectively, where $p_i+q_i = 1$ and $p_ia_i+q_ib_i = 0$. The non-random environmental effects are due to individuals sharing a common environment, $u$ being assumed to take the same value for full sibs and $c$ the same value for all individuals housed together, and either or both may be assumed zero as appropriate. In the cases considered below, $c$ takes the same value for all individuals full or half sib to each other and of the same generation. Genetic value, $Y$, is given by—

$$Y = M+g_1+g'_1+g_2+g'_2+\ldots+g_k+g'_k$$

and all random variables in the model are of mean zero and uncorrelated (except as below) in accordance with the assumptions previously adopted.

Using the term locus to refer to a specific section of a chromosome, two loci may be defined as identical if they have both been derived from some one locus at a previous meiotic division, so that, excepting mutation, the same allele of a factor is represented by the genes at those loci. Then, following Malécot (1948), one may define the coefficient of inbreeding (c.i.), $f$, of an individual as the probability that the two loci representing any factor are identical, and the coefficient of relationship (c.r.), $g$, of two individuals as the probability that two loci representing any factor, one being nominated at random from each individual, are likewise identical. Thus, the coefficient of inbreeding of an individual is the coefficient of relationship of its parents, and the population to which the probabilities refer is hypothetical and generated by infinite repetitions of the pattern of matings antecedent to the individuals being considered. For non-inbred individuals, the c.r. of an individual $A$ with itself is $\frac{1}{2}$; of $A$ and $A$'s full sib $\frac{1}{4}$; of $A$ and $A$'s progeny $\frac{1}{4}$; of $A$ and $A$'s half sib $\frac{1}{8}$; of $A$'s full sib and $A$'s progeny $\frac{1}{8}$; and of $A$'s half sib and $A$'s progeny $\frac{1}{16}$.

Then, if an individual has a c.i. $f$, expected values are such that $E(g_ig'_i) = f.\ E(g_i)^2 = f.\ E(g'_i)^2$, and, similarly, if the c.r. of two

individuals is $g$ (barred symbols indicating the second individual),

$$E(g_i\bar{g}_i) = E(g_i\bar{g}'_i) = E(g'_i\bar{g}_i) = E(g'_i\bar{g}'_i) = g.\ E(g_i)^2 = g.\ E(\bar{g}_i)^2.$$

Define $V_G = 2\overset{k}{\Sigma}E(g_i)^2$, $V_U = E(u)^2$, $V_C = E(c)^2$, $V_E = E(e)^2$, $V = V_G+V_U+V_C+V_E$ and $h^2 = V_G/V$, $u^2 = V_U/V$, $c^2 = V_C/V$, $e^2 = V_E/V$, so that $h^2+u^2+c^2+e^2 = 1$. The heritability of the character being considered is $h^2$ and the reproductibility $(h^2+u^2+c^2)$.

## 3. ESTIMATION FORMULAE

The genetic value $Y$ of an individual is to be estimated from the values of some selection of the means $y$, $\bar{F}$, $\bar{H}$, $\bar{P}$, $\bar{F}'$, $\bar{S}$ and $\bar{S}'$, and the appropriate formulae are listed below for a variety of cases, being set out as :—

(a) The means on which the estimate is to be based.

(b) The corresponding simple or partial regression coefficients.

(c) The variance of $Y$ accounted for by the introduction of successive means in the order indicated. The relative selective advance is the square root of the sum of these items divided by $h^2$.

These formulae provide, in effect, average values over a population defined by the partition of the variance of phenotypic value as $h^2+u^2+c^2+e^2 = 1$. Define $T = (\frac{1}{2}h^2+u^2+c^2)$, $\bar{T} = (\frac{1}{4}h^2+c^2)$, $k = (\overset{m}{\Sigma}n^2_i)/m\bar{n}^2$ and $l = (\overset{p}{\Sigma}q^2_i)/p\bar{q}^2$. Then—

1. (a) $y$.

   (b) $b_{Yy}$, $h^2$

   (c) $y$, $h^4$

2. (a) $\bar{F}$.

   (b) $b_{Y\bar{F}}$, $\dfrac{nh^2}{2[1+(n-1)T]}$

   (c) $\bar{F}$, $\dfrac{nh^4}{4[1+(n-1)T]}$

3. (a) $\bar{H}$.

   (b) $b_{Y\bar{H}}$, $\dfrac{m\bar{n}h^2}{4[1+(k\bar{n}-1)T+\bar{n}(m-k)\bar{T}]}$

   (c) $\bar{H}$, $\dfrac{m\bar{n}h^4}{16[1+(k\bar{n}-1)T+\bar{n}(m-k)\bar{T}]}$

4. (a) $\bar{P}$.

(b) $b_{Y\bar{P}}$, $\dfrac{p\bar{q}h^2}{2[1+(l\bar{q}-1)T+\bar{q}(p-l)\bar{T}]}$

(c) $\bar{P}$, $\dfrac{p\bar{q}h^4}{4[1+(l\bar{q}-1)T+\bar{q}(p-l)\bar{T}]}$

5. (a) $\bar{F}'$.

(b) $b_{Y\bar{F}'}$, $\dfrac{(n+2)h^2}{2(1+nT)}$

(c) $\bar{F}'$, $\dfrac{(n+2)^2h^4}{4(n+1)(1+nT)}$

6. (a) $\bar{S}$.

(b) Define $m' = (m+1)$, $\bar{n}' = (n+m\bar{n})/(m+1)$,
$k' = (m+1)(n^2+m\bar{n}^2k)/(n+m\bar{n})^2$

$b_{Y\bar{S}}$, $\dfrac{(n+m'\bar{n}')h^2}{4[1+(k'\bar{n}'-1)T+\bar{n}'(m'-k')\bar{T}]}$

(c) $\bar{S}$, $\dfrac{(n+m'\bar{n}')^2h^4}{16m'\bar{n}'[1+(k'\bar{n}'-1)T+\bar{n}'(m'-k')\bar{T}]}$

7. (a) $\bar{S}'$.

(b) Define $m' = (m+1)$, $\bar{n}' = (1+n+m\bar{n})/(m+1)$,
$k' = (m+1)[(n+1)^2+m\bar{n}^2k]/(1+n+m\bar{n})^2$

$b_{Y\bar{S}'}$, $\dfrac{(3+n+m'\bar{n}')h^2}{4[1+(k'\bar{n}'-1)T+\bar{n}'(m'-k')\bar{T}]}$

(c) $\bar{S}'$, $\dfrac{(3+n+m'\bar{n}')^2h^4}{16m'\bar{n}'[1+(k'\bar{n}'-1)T+\bar{n}'(m'-k')\bar{T}]}$

8. (a) $y$, $\bar{F}$.

(b) $b_{Y\bar{F}.y}$, $\dfrac{n(1-2T)h^2}{2(1-T)(1+nT)}$

$b_{Xy.\bar{F}}$, $h^2-Tb_{Y\bar{F}.y}$

(c) $y$, $h^4$

$\bar{F}$, $\dfrac{n(1-2T)^2h^4}{4(1-T)(1+nT)}$

9. (a) $y$, $\bar{H}$.

(b) $b_{Y\bar{H}.y}$, $\dfrac{m\bar{n}(1-4\bar{T})h^2}{4[1+(k\bar{n}-1)T+\bar{n}(m-k)\bar{T}-m\bar{n}\bar{T}^2]}$

$b_{Xy.\bar{H}}$, $h^2-\bar{T}b_{Y\bar{H}.y}$

(c) $y$, $h^4$

$\bar{H}$, $\dfrac{m\bar{n}(1-4\bar{T})^2h^4}{16[1+(k\bar{n}-1)T+\bar{n}(m-k)\bar{T}-m\bar{n}\bar{T}^2]}$

10. (a) $y$, $\bar{P}$.

(b) $b_{Y\bar{P}.y}$, $\dfrac{2p\bar{q}(1-h^2)h^2}{\{4[1+(l\bar{q}-1)T+\bar{q}(p-l)\bar{T}]-p\bar{q}h^4\}}$

$b_{Yy.\bar{P}}$, $(1-\frac{1}{2}b_{Y\bar{P}.y})h^2$

(c) $y$, $h^4$

$\bar{P}$ $\dfrac{p\bar{q}(1-h^2)^2h^4}{\{4[1+(l\bar{q}-1)T+\bar{q}(p-l)\bar{T}]-p\bar{q}h^4\}}$

11. (a) $\bar{F}$, $\bar{P}$.

(b) $b_{Y\bar{P}.\bar{F}}$, $\dfrac{2p\bar{q}\{4[1+(n-1)T]-nh^2\}h^2}{\{16[1+(n-1)T][1+(l\bar{q}-1)T+\bar{q}(p-l)\bar{T}]-np\bar{q}h^4\}}$

$b_{Y\bar{F}.\bar{P}}$, $\dfrac{n}{2[1+(n-1)\bar{T}]}(1-\frac{1}{2}b_{Y\bar{P}.\bar{F}})h^2$

(c) $\bar{F}$, $\dfrac{nh^4}{4[1+(n-1)T]}$

$\bar{P}$, $\dfrac{p\bar{q}\{4[1+(n-1)T]-nh^2\}^2h^4}{4[1+(n-1)T]\{16[1+(n-1)T][1+(l\bar{q}-1)T+\bar{q}(p-l)\bar{T}]-np\bar{q}h^4\}}$

12. (a) $\bar{F}$, $\bar{H}$.

(b) $b_{Y\bar{H}.\bar{F}}$, $\dfrac{m\bar{n}[1+(n-1)T-2n\bar{T}]h^2}{4\{[1+(n-1)T][1+(k\bar{n}-1)T+\bar{n}(m-k)\bar{T}]-m\bar{n}n\bar{T}^2\}}$

$b_{Y\bar{F}.\bar{H}}$, $\dfrac{n}{2[1+(n-1)T]}(h^2-2\bar{T}\,b_{Y\bar{H}.\bar{F}})$

(c) $\bar{F}$, $\dfrac{nh^4}{4[1+(n-1)T]}$

$\bar{H}$, $\dfrac{m\bar{n}[1+(n-1)T-2n\bar{T}]^2h^4}{16[1+(n-1)T]\{[1+(n-1)T][1+(k\bar{n}-1)T+\bar{n}(m-k)\bar{T}]-m\bar{n}n\bar{T}^2\}}$

13. (a) $\bar{H}$, $\bar{P}$. (where $n_i = 1$ for all $i$).

(b) $b_{Y\bar{P}.\bar{H}}$, $\dfrac{2p\bar{q}\{16[1+(m-1)\bar{T}]-mh^2\}h^2}{\{64[1+(m-1)\bar{T}][1+(l\bar{q}-1)T+\bar{q}(p-l)\bar{T}]-mp\bar{q}h^4\}}$

$b_{Y\bar{H}.\bar{P}}$, $\dfrac{m}{4[1+(m-1)\bar{T}]}(1-\frac{1}{2}b_{Y\bar{P}.\bar{H}})h^2$

(c) $\bar{H}$, $\dfrac{mh^4}{16[1+(m-1)\bar{T}]}$

$P$, $\dfrac{p\bar{q}\{16[1+(m-1)\bar{T}]-mh^2\}^2h^4}{16[1+(m-1)\bar{T}]\{64[1+(m-1)\bar{T}][1+(l\bar{q}-1)T+\bar{q}(p-l)\bar{T}]-mp\bar{q}h^4\}}$

14. (a) $y$, $F$, $H$.

(b) $b_{YH.yF}$, $\dfrac{m\bar{n}[1+nT-2(n+2)\bar{T}]h^2}{4\{(1+nT)[1+(k\bar{n}-1)T+\bar{n}(m-k)\bar{T}]-m\bar{n}(n+1)\bar{T}^2\}}$

$b_{YF.yH}$, $\dfrac{n(1-2T)h^2}{2(1-T)(1+nT)}-\dfrac{n\bar{T}}{(1+nT)}\,b_{YH.yF}$

$b_{Yy.FH}$, $(h^2-Tb_{YF.yH}-\bar{T}b_{YH.yF})$

(c) $y$, $h^4$

$F$, $\dfrac{n(1-2T)^2h^4}{4(1-T)(1+nT)}$

$H$, $\dfrac{m\bar{n}[1+nT-2(n+2)\bar{T}]^2h^4}{16(1+nT)\{(1+nT)[1+(k\bar{n}-1)T+\bar{n}(m-k)\bar{T}]-m\bar{n}(n+1)\bar{T}^2\}}$

This case is considered by Osborne (1957), and substituting $(n-1)$ for $n$, $(d-1)$ for $m$, $n$ for $\bar{n}$ and 1 for $k$,

$$b_{YH.yF}=\frac{n(d-1)[1+(n-1)T-2(n+1)\bar{T}]h^2}{4[1+(n-1)T-n\bar{T}][1+(n-1)T+n(d-1)\bar{T}]}$$

$$b_{YF.yH}=\frac{(n-1)(1-2T)h^2}{2(1-T)[1+(n-1)T]}-\frac{(n-1)\bar{T}}{[1+(n-1)T]}\,b_{YH.yF}$$

$$b_{Yy.FH}=(h^2-Tb_{YF.yH}-\bar{T}b_{YH.yF})$$

and Osborne's coefficients $b_2$ and $b_3$ are then given by—

$$b_2=\frac{n(d-1)b_{YF.yH}-(n-1)b_{YH.yF}}{(d-1)[(n-1)b_{Yy.FH}-b_{YF.yH}]}$$

$$b_3=\frac{d(n-1)b_{YH.yF}}{(d-1)[(n-1)b_{Yy.FH}-b_{YF.yH}]}$$

15. (a) $y$, $F$, $P$.

(b) $b_{YF.yF}$, $\dfrac{2p\bar{q}\{4(1-T)(1+nT)-(4+n-4T)h^2\}h^2}{\{16(1-T)(1+nT)[1+(l\bar{q}-1)T+\bar{q}(p-l)\bar{T}]-p\bar{q}(4+n-4T)h^4\}}$

$b_{YF.yH}$, $\dfrac{n(1-2T)}{2(1-T)(1+nT)}\,(1-\frac{1}{2}b_{YF.yF})h^2$

$b_{Yy.FH}$, $(h^2-Tb_{YF.yH}-\frac{1}{2}h^2b_{YF.yF})$

(c) $y$, $h^4$

$F$, $\dfrac{n(1-2T)^2h^4}{4(1-T)(1+nT)}$

$P$, $\dfrac{p\bar{q}\{4(1-T)(1+nT)-(4+n-4T)h^2\}^2h^4}{4(1-T)(1+nT)\{16(1-T)(1+nT)[1+(l\bar{q}-1)T+\bar{q}(p-l)\bar{T}]-p\bar{q}(4+n-4T)h^4\}}$

16. (a) $y$, $\bar{H}$, $\bar{P}$ (where $n_i = 1$ for all $i$)

(b) $b_{Y\bar{B}.y_B}$, $\dfrac{2p\bar{q}\{16(1-\bar{T})(1+m\bar{T})-[16+m+8(m-2)\bar{T}]h^2\}h^2}{\{64(1-\bar{T})(1+m\bar{T})[1+(l\bar{q}-1)\bar{T}+\bar{q}(p-l)\bar{T}]-p\bar{q}[16+m+8(m-2)\bar{T}]h^4\}}$

$b_{Y\bar{B}.y_{\bar{B}}}$, $\dfrac{m(1-4\bar{T})}{4(1-\bar{T})(1+m\bar{T})}(1-\frac{1}{2}b_{Y\bar{B}.y_B})h^2$

$b_{\bar{Y}y.B\bar{B}}$, $(h^2-\bar{T}b_{Y\bar{B}.y_{\bar{B}}}-\frac{1}{2}h^2b_{Y\bar{B}.y_B})$

(c) $y$, $h^4$

$\bar{H}$, $\dfrac{m(1-4\bar{T})^2h^4}{16(1-\bar{T})(1+m\bar{T})}$

$\bar{P}$, $\dfrac{p\bar{q}\{16(1-\bar{T})(1+m\bar{T})-[16+m+8(m-2)\bar{T}]h^2\}^2h^4}{16(1-\bar{T})(1+m\bar{T})\{64(1-\bar{T})(1+m\bar{T})[1+(l\bar{q}-1)\bar{T}+\bar{q}(p-l)\bar{T}]-p\bar{q}[16+m+8(m-2)\bar{T}]h^4\}}$

## 4. INBREEDING

The formulae of the preceding section can all be generalised so as to apply to inbred individuals, the necessary coefficients of inbreeding and relationship being readily calculable, given the individuals on which the estimation is to be based and the pattern of matings which gave rise to them.

Consider, as an example, the random mating of a finite set of $M$ males and $F$ females where the coefficients of inbreeding and relationship in successive generations are $f'$, $g'$ and $f$, $g$, respectively. Then, if $H$ is the harmonic mean of $M$ and $F$, *i.e.* $2MF/(M+F)$,

$$f = g',\ g = \frac{1}{H}\left[\tfrac{1}{4}+\tfrac{1}{4}f'+\tfrac{1}{2}(2H-1)g'\right],$$

and the coefficient of relationship between individuals of the male (female) parent and progeny groups, $g_M$ ($g_F$), is given by substituting $M$ ($F$) for $H$ in the above formula for $g$. This recurrence relation is also applicable to other mating systems if appropriate values are given to $M$ and $F$. Thus for selfing $M = F = \frac{1}{2}$, $H = \frac{1}{2}$; for full sib mating $M = F = 1$, $H = 1$; for half sib mating $M = 1$, $F = \infty$, $H = 2$; and for random mating (infinite population) $M = F = \infty$, $H = \infty$. Such coefficients may be calculated successively from an initial stage when individuals are non-inbred and unrelated, and are applicable in such formulæ as are given in the next section.

Moreover, when pedigrees are known precisely, it may be noted that, while the coefficients of inbreeding and relationship of specific individuals refer (as previously stated) to the probability of certain loci being identical, it is, of course, a matter of chance, subject to such probabilities, whether those loci are or are not actually identical. This latter statement is equally true for the random mating considered

above, where pedigrees are not precisely known (any two individuals may actually have 0, 1 or 2 parents in common), but now the coefficients do not give the probability of certain loci being identical for the specific individuals concerned, but the average of such values as are generated by the random choice of parents for those individuals.

## 5. ESTIMATION FORMULAE—INDIVIDUALS INBRED

Consider an individual $A$ and the groups of relatives specified in section 1. In $A$'s generation the c.i. and c.r. within full sib groups are $f'$ and $g'$, respectively, and the c.r. between full sib groups, $\bar{g}'$. Similarly, in the progeny generation the c.i. and c.r. within full sib groups are $f$ and $g$, respectively, and the c.r. between full sib groups, $\bar{g}$. In addition, the c.r. between $A$'s progeny and $A$ is $G$; $A$'s progeny and $A$'s full sibs, $\bar{G}$; and $A$'s progeny and $A$'s half sibs, $\bar{G}'$.

Define $S' = (1+f'h^2)$, $S = (1+fh^2)$, $T' = (2g'h^2+u^2+c^2)$, $T = (2gh^2+u^2+c^2)$, $\bar{T}' = (2\bar{g}'h^2+c^2)$, $\bar{T} = (2\bar{g}h^2+c^2)$ and all other symbols as before. When individuals are non-inbred, $f' = 0$, $g' = \frac{1}{4}$, $\bar{g}' = \frac{1}{8}$, $f = 0$, $g = \frac{1}{4}$, $\bar{g} = \frac{1}{8}$, $G = \frac{1}{4}$, $\bar{G} = \frac{1}{8}$, $\bar{G}' = \frac{1}{16}$, as previously stated, and $S' = S = 1$, $T' = T$, $\bar{T}' = \bar{T}$. The following variances and covariances for the general case may now be derived :—

$$V(Y) = (1+f')h^2$$

$$V(y) = S'$$

$$V(\bar{F}) = \frac{S'+(n-1)T'}{n}$$

$$V(\bar{H}) = \frac{S'+(k\bar{n}-1)T'+\bar{n}(m-k)\bar{T}'}{m\bar{n}}$$

$$V(\bar{P}) = \frac{S+(l\bar{q}-1)T+\bar{q}(p-l)\bar{T}}{p\bar{q}}$$

$$V(F') = \frac{S'+nT'}{(n+1)}$$

$V(\bar{S})$ as for $V(\bar{H})$ substituting $m'$, $\bar{n}'$, $k'$ for $m$, $\bar{n}$, $k$ where

$$m' = (m+1),\ \bar{n}' = \frac{(n+m\bar{n})}{(m+1)},$$

$$k' = \frac{(m+1)(n^2+m\bar{n}^2k)}{(n+m\bar{n})^2}$$

$V(\bar{S}')$ as for $V(\bar{H})$ substituting $m'$, $\bar{n}'$, $k'$ for $m$, $\bar{n}$, $k$ where

$$m' = (m+1),\ \bar{n}' = \frac{(1+n+m\bar{n})}{(m+1)},$$

$$k' = \frac{(m+1)[(n+1)^2+m\bar{n}^2k]}{(1+n+m\bar{n})^2}$$

$$W(\Upsilon y) = (1+f')h^2$$
$$W(\Upsilon \bar{F}) = 2g'h^2$$
$$W(\Upsilon \bar{H}) = 2\bar{g}'h^2$$
$$W(\Upsilon \bar{P}) = 2Gh^2$$
$$W(\Upsilon \bar{F}') = \frac{[1+f'+2ng']h^2}{(n+1)}$$
$$W(\Upsilon \bar{S}) = \frac{2[n(g'-\bar{g}')+m'\bar{n}'\bar{g}']h^2}{(n+m\bar{n})}$$
$$W(\Upsilon \bar{S}') = \frac{[1+(f'-2\bar{g}')+2n(g'-\bar{g}')+2m'\bar{n}'\bar{g}']h^2}{(1+n+m\bar{n})}$$
$$W(y\bar{F}) = T'$$
$$W(y\bar{H}) = \bar{T}'$$
$$W(y\bar{P}) = 2Gh^2$$
$$W(\bar{F}\bar{H}) = \bar{T}'$$
$$W(\bar{F}\bar{P}) = 2\bar{G}h^2$$
$$W(\bar{H}\bar{P}) = 2\bar{G}'h^2$$

The regression analysis of $\Upsilon$ for any selection of the independent variables may now be carried through numerically, for any particular case, by the use of these formulae or, alternatively, the regression formulae calculated explicitly (as previously and when not inordinately long), numerical values being substituted later. Although these more general formulae do not appear to be particularly useful, a selection of the simpler cases is given below and set out as before. The relative selective advance is now the square root of the sum of the items listed under (c) divided by $(1+f')h^2/\sqrt{S'}$, rather than $h^2$ as before.

1. (a) $y$

   (b) $b_{xy}$, $\dfrac{(1+f')h^2}{S'}$

   (c) $y$, $\dfrac{(1+f')^2h^4}{S'}$

2. (a) $\bar{F}$

   (b) $b_{x\bar{F}}$, $\dfrac{2ng'h^2}{S'+(n-1)T'}$

   (c) $\bar{F}$, $\dfrac{4n(g')^2h^4}{S'+(n-1)T'}$

3. (a) $\bar{F}'$.

(b) $b_{Y\bar{F}'}$, $\dfrac{(1+f'+2ng')h^2}{S'+nT'}$

(c) $\bar{F}'$, $\dfrac{(1+f'+2ng')^2h^4}{(n+1)(S'+nT')}$

4. (a) $\bar{P}$.

(b) $b_{Y\bar{P}}$, $\dfrac{2p\bar{q}Gh^2}{[S+(l\bar{q}-1)T+\bar{q}(p-l)\bar{T}]}$

(c) $\bar{P}$, $\dfrac{4p\bar{q}G^2h^4}{[S+(l\bar{q}-1)T+\bar{q}(p-l)\bar{T}]}$

5. (a) $y$, $\bar{F}$.

(b) $b_{Y\bar{F}.y}$, $\dfrac{n[2g'S'-(1+f')T']h^2}{(S'-T')(S'+nT')}$

$b_{Yy.\bar{F}}$, $\dfrac{(1+f')h^2}{S'} - \dfrac{T'}{S'}\, b_{Y\bar{F}.y}$

(c) $y$, $\dfrac{(1+f')^2h^4}{S'}$

$\bar{F}$, $\dfrac{n[2g'S'-(1+f')T']^2h^4}{S'(S'-T')(S'+nT')}$

6. (a) $y$, $\bar{P}$. (where $p = 1$, $\bar{q} = q$)

(b) $b_{Y\bar{P}.y}$, $\dfrac{2qG(1-h^2)h^2}{\{S'[S+(q-1)T]-4qG^2h^4\}}$

$b_{Yy.\bar{P}}$, $\dfrac{(1+f')h^2}{S'} - \dfrac{2Gh^2}{S'}\, b_{Y\bar{P}.y}$

(c) $y$, $\dfrac{(1+f')^2h^4}{S'}$

$\bar{P}$, $\dfrac{4qG^2(1-h^2)^2h^4}{S'\{S'[S+(q-1)T]-4qG^2h^4\}}$

7. (a) $\bar{F}$, $\bar{P}$. (where $p = 1$, $\bar{q} = q$)

(b) $b_{Y\bar{P}.\bar{F}}$, $\dfrac{2q\{G[S'+(n-1)T']-2n\bar{G}g'h^2\}h^2}{\{[S'+(n-1)T'][S+(q-1)T]-4nq\bar{G}^2h^4\}}$

$b_{Y\bar{P}.\bar{F}}$, $\dfrac{2n}{[S'+(n-1)T']}\,(g'-\bar{G}b_{Y\bar{P}.\bar{F}})h^2$

(c) $\bar{F}$, $\dfrac{4n(g')^2h^4}{[S'+(n-1)T']}$

$\bar{P}$, $\dfrac{4q\{G[S'+(n-1)T']-2n\bar{G}g'h^2\}^2h^4}{[S'+(n-1)T']\{[S'+(n-1)T'][S+(q-1)T]-4nq\bar{G}^2h^4\}}$

## 6. DIFFERENCES IN GENETIC VALUE

When related individuals are to be compared, the foregoing formulæ require modification, and several cases are discussed below.

### (i) *Sib tests*

Consider two groups of individuals, $A$ and $A$'s $m$ associates and $B$ and $B$'s $n$ associates. The c.i. and c.r. within the groups are $f'$ and $g'$, respectively, and the c.r. between the groups is $\bar{g}'$. The fraction of phenotypic variance due to common environment within the group is $u^2$.

(a) $(Y_A - Y_B)$ is to be estimated from either $(y_A - y_B)$ or $(\bar{F}_A - \bar{F}_B)$ or both jointly. Defining $K = 2mn/(m+n)$, the regression formulae required are those given as 1, 2 and 5 of section 5 when $(f' - 2\bar{g}')$ is substituted for $f'$, $(g' - \bar{g}')$ for $g'$, $K$ for $n$ and with $c^2 = 0$.

(b) $(Y_A - Y_B)$ is to be estimated from $(\bar{F}'_A - \bar{F}'_B)$. The regression formulae required are those given as 3 of section 5 where $K'$ is substituted for $(n+1)$ [$K'$ being $2(m+1)(n+1)/(m+n+2)$], $(f' - 2\bar{g}')$ and $(g' - \bar{g}')$ replacing $f'$ and $g'$ as above and with $c^2 = 0$.

### (ii) *Progeny tests*

Consider an individual $A$ together with $A$'s $p$ progeny and an individual $B$ together with $B$'s $q$ progeny. The c.i. and c.r. within the progeny groups are $f$ and $g$, respectively, and the c.r. between the groups is $\bar{g}$. In addition, the c.r. of $A$ and $A$'s progeny (likewise, $B$ and $B$'s progeny) is $G$, while the c.r. of $A$ and $B$'s progeny (likewise, $B$ and $A$'s progeny) is $G_0$. The fraction of phenotypic variance due to common environment within the groups is again $u^2$.

$(Y_A - Y_B)$ is to be estimated from $(\bar{P}_A - \bar{P}_B)$. Defining $L = 2pq/(p+q)$, the regression formulae required are those given as 4 of section 5 (for the case where $p = 1$, $\bar{q} = q$) when $(f - 2\bar{g})$ is substituted for $f$, $(g - \bar{g})$ for $g$, $(G - G_0)$ for $G$, $L$ for $q$ and with $c^2 = 0$.

## 7. DISCUSSION

The greatest scope for the use of formulae such as have been considered in the previous sections is in the field of poultry breeding and, although it is not intended to discuss alternative breeding plans here, several points mentioned in the introduction, and referring to this application, may be considered briefly.

Firstly, as a matter of interest, the use of simple instead of partial regression coefficients may be examined, for Lerner's example and numerical values are given in table 1. The actual gain was 5·6 eggs, the estimate using simple regression coefficients 5·28 eggs, and that using partial regression coefficients 4·65 eggs. This last figure is 17 per cent. low on the observed value rather than 5·7 per cent. as

indicated by the former estimate, and the difference is therefore not trivial.

Secondly, as regards Osborne's recommendation as to the use of half sib data, it is of interest to note the influence of non-random

TABLE 1

*Regression coefficients of* Y *on* y, $\bar{F}$, *and* $\bar{P}$ ($h^2 = 0{\cdot}045$)

| Independent variable | Sire | | | Dam | | |
|---|---|---|---|---|---|---|
| | No. of individuals | Simple | Partial | No. of individuals | Simple | Partial |
| $y$ | ... | ... | ... | 1 | 0·045 | 0·039 |
| $\bar{F}$ | 8 | 0·155 | 0·122 | 7 | 0·138 | 0·124 |
| $\bar{P}$ | 36 | 0·452 | 0·425 | 8 | 0·155 | 0·140 |

environmental effects on relative selective advance. Numerical values are given in table 2 for Osborne.s example (in the notation of this paper, $n = 5$, $m = 15$, $\bar{n} = 6$, $h^2 = 0{\cdot}05$, $u^2 = 0$), several values being adopted for $c^2$. It is evident that when heritability is low a quite small non-random environmental component may render the use of half

TABLE 2

*Relative selective advance*

($n = 5$, $m = 15$, $n = 6$, $h^2 = 0{\cdot}05$, $u^2 = 0$)

| Estimation based on | $c^2$ | | |
|---|---|---|---|
| | 0 | 0·025 | 0·05 |
| $\bar{F}'$ . . . . | 1·35 | 1·28 | 1·22 |
| $S'$ . . . . | 1·77 | 1·23 | 1·00 |
| $y$ and $\bar{F}$ . . . | 1·42 | 1·36 | 1·31 |
| $y$, $\bar{F}$ and $\bar{H}$ . . . | 1·98 | 1·53 | 1·36 |

sib data undesirable, and (although Osborne is well aware of this) it is suggested that such data should only be used if positive steps are taken to ensure that such effects are zero and that no *a priori* assumption of their being so is either adequate or justifiable.

Finally, it may be noted that $k$ and $l$ (which are a measure of the inequality in half sib and progeny family sizes) occur in the denominators of the formulae for the relative selective advance and, being always greater than or equal to 1, maximum advance is attained in this latter case when half sib and progeny families are all of equal size. However, as Osborne suggests, inequality in this respect appears to be trivial.

## 8. SUMMARY

Explicit formulae are presented for the regression coefficients, simple or partial, of the genetic value of an individual on the various mean phenotypic values of its relatives, and for the relative selective advance from the use of those means, these formulae being derived on the assumption that phenotypic value is due to independent additive factors with random and non-random, non-interacting environmental effects. The necessary formulae are also provided for extension to those cases where individuals are inbred, and the estimation of differences in genetic value is discussed for several simple situations.

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# THE LUTHERAN-SECRETOR LINKAGE IN MAN: SUPPORT FOR MOHR'S FINDINGS

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THE first autosomal linkage to be recognised in Man was that between the Lutheran genes and genes responsible for the Lewis phenotype of red cells (Mohr, 1951*a*, 1951*b*, 1954, 1956). Owing presumably to the rarity of informative matings no confirmation of the linkage has appeared from other laboratories. When we last analysed our own family data for linkage (Race and Sanger, 1954) we had practically nothing to contribute to the Lutheran-Lewis comparison, but since that time two very informative families have been encountered. Our collected results now bring strong support for the linkage which, in the light of our present understanding of the Lewis phenotypes, now appears to be between the Lutheran and secretor genes—a possibility that had been appreciated by Mohr.

## 1. GENES

### (i) *The Lutheran genes*

Nothing complicated is yet known about the Lutheran genes: $Lu^a$ in single dose or in double dose causes the presence on the red cells of the antigen $Lu^a$. The antigen $Lu^b$, and therefore the gene $Lu^b$, was shown to have a positive existence when, in 1956, Cutbush and Chanarin discovered the antibody anti-$Lu^b$.

### (ii) *The secretor genes*

The presence or absence of water-soluble ABH substance in the saliva still appears relatively straightforward. The secretor genes, *Se se*, so modify the action of the *ABO* genes that when the genotype is *se se* practically no ABH substance is to be found in the saliva, whereas large quantities of the appropriate substances are present when the genotype is *Se Se* or *Se se*. (The antigen H is not understood; it is certainly not the product of the gene *O*, but its presence or absence in the saliva of people of group O serves to classify them as secretors or non-secretors.) The words secretor and non-secretor refer to the presence or absence of the ABH substances alone; they should not be used for the presence or absence of other substances—such as $Le^a$ which has a quite different genetic background.

### (iii) *The Lewis genes*

The inheritance of the Lewis antigens is, on the other hand, most complex. Our present views are based mainly on the work of Grubb (1948, 1950, 1951), Ceppellini (1955*a*, 1955*b*), Ceppellini and Siniscalco (1955), Sneath and Sneath (1955), and Mäkelä and Mäk.ä (1956).

The antigen Le$^a$ is thought to be primarily an antigen of the body fluids, notably of the saliva, which only secondarily becomes hitched onto the red cells (Grubb, 1950; Ceppellini, 1955*a*, 1955*b*; Sneath

TABLE 1

*Scheme of the Lewis system and secretion of ABH*

(Slightly modified, from Grubb, 1951 and Ceppellini, 1955)

| Genotypes | Phenotypes | | |
|---|---|---|---|
| | Saliva | | Red cells |
| | ABH | Le$^a$ | |
| *Se Se LL*<br>*Se Se Ll*<br>*Se se LL*<br>*Se se Ll* | + | + | Le(a−b+) |
| *se se LL*<br>*se se Ll* | − | + | Le(a+b−) |
| *Se Se ll*<br>*Se se ll* | + | − | Le(a−b−) |
| *se se ll* | − | − | Le(a−b−) |

and Sneath, 1955). The gene which produces the antigen has been called *L*; it is dominant in its effect and is not linked to the secretor or *ABO* genes (Ceppellini, 1955*a*; Ceppellini and Siniscalco, 1955).

The Lewis phenotype of the red cells is thought to be the result of interaction between the *Ll* genes and the secretor genes as shown in table 1. It seems that there is a limited amount of substrate that can be made into water-soluble ABH substance or Le$^a$ substance. The demands of the *A*, *B* and *O* and secretor genes are satisfied first and, consequently, in secretors but little substrate is left for the *L* gene to mould into Le$^a$ substance: some Le$^a$ substance does reach the saliva but not enough reaches the plasma to hook onto the red cells and make them give the Le(a+) reaction. When the *ABO* and secretor genes require none of this substrate (that is in non-secretors) all of it is available for moulding into Le$^a$ substance by the gene *L*; then the saliva is saturated with Le$^a$ substance and enough reaches the plasma to hook onto the red cells and make them give the Le(a+) reaction.

There does not seem to be an *Le*$^b$ gene, and the red cell antigen Le$^b$ appears to be a product of the interaction of the *ABO* genes, the secretor gene *Se* and the gene *L*. A further complication is that the reactions of anti-Le$^b$ sera are influenced by the ABO group of the red cells : using most anti-Le$^b$ sera too many $A_1$ and $A_1B$ red cells are classified as Le(b−).

Nevertheless, as a result of the interaction of the Lewis and secretor genes the red cell phenotype Le(a+) behaves like a recessive character for it reflects the presence of two *se* genes and the following relations hold :

Le(a+b−) red cells belong to non-secretors,
Le(a−b+) red cells belong to secretors,
Le(a−b−) red cells usually, but not always, belong to secretors.

Consequently when no saliva has been available we can confidently assume that all people whose red cells give the reaction Le(a+) are non-secretors and that those whose red cells give the reaction Le(a−b+) are secretors. We would usually be right in assuming that the 6 per cent. or so of white people whose red cells give the reaction Le(a−b−) are secretors.

## 2. LINKAGE DATA

Table 2 contains all the families of two or more children whose red cells we have tested with ant-Lu$^a$ and anti-Le$^a$ and in which a parent is Lu(a+). When samples of saliva were available they have been classified as secretor or non-secretor ; most of them have been further tested for Le$^a$ substance.

In the analysis the *u* statistics of Fisher (1935) have been used as elaborated by Finney (1940). Only " certain " families have been scored and they have been treated as Finney's Mating Types 13 and 14 (in his table 1). Finney's symbols have been translated thus : W = Lu(a+) and w = Lu(a−) ; T = secretor, Le$^a$ present in saliva (L), or Le(a−) phenotype of red cells, t = non-secretor, Le$^a$ absent from saliva (ll), or Le(a+) phenotype of red cells, according to the characters being analysed. In one comparison, that between secretor and L, Mating Type 15 has been used.

The results of the various comparisons are given in table 3. The last column but one shows the significance of the results : Finney states that if $\Sigma(\lambda)$ exceeds $2{\cdot}33\sqrt{\Sigma(\kappa)}$ linkage is established at the 1 in 100 level of probability. Linkage having been established, the last column shows the estimate of the recombination fraction which, again according to Finney, is $\frac{1}{2}\left[1-\sqrt{\frac{\Sigma(\lambda)}{\Sigma(\kappa)}}\right]$.

In table 4 the latest results of Mohr (1954, 1956) and of Ceppellini and Siniscalco (1955) are added to our own.

TABLE 2—*The families*

| No. | Father | Mother | Children | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | 1 | 2 | 3 | 4 | 5 | 6 | 7 |
| 1 | Lu(a−)<br>non-sec.<br>Le(a+b−)<br>L<br>$A_1$ | Lu(a+)<br>sec.<br>Le(a−b+)<br>L<br>O | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>$A_1$ | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>$A_1$ | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>O | Lu(a+)<br>non-sec.<br>Le(a+b−)<br>L<br>O | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>$A_1$ | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>O | Lu(a+)<br>sec.<br>Le(a−b+)<br>L<br>$A_1$ |
| 2 | Lu(a−b+)<br>non-sec.<br>Le(a+b−)<br>L<br>O | Lu(a+)<br>sec.<br>Le(a−b+)<br>L<br>O | Lu(a+b+)<br>non-sec.<br>Le(a+b−)<br>L<br>O | Lu(a+b+)<br>non-sec.<br>Le(a+b−)<br>L<br>O | Lu(a+b+)<br>non-sec.<br>Le(a+b−)<br>L<br>O | Lu(a−b+)<br>sec.<br>Le(a−b+)<br>L<br>O | Lu(a+b+)<br>non-sec.<br>Le(a+b−)<br>L<br>O | | |
| 3 | Lu(a+)<br>sec.<br>Le(a−b−)<br>ll<br>O | Lu(a−)<br>non-sec.<br>Le(a+b−)<br>L<br>$A_1$ | Lu(a+)<br>Le(a−b+)<br>O | Lu(a−)<br>non-sec.<br>Le(a+b−)<br>L<br>O | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>O | | | | |
| 4 | Lu(a+)<br>Le(a−b+)<br>O | Lu(a−)<br>Le(a+b−)<br>$A_1$ | Lu(a+)<br>Le(a−b+)<br>$A_1$ | Lu(a−)<br>Le(a+b−)<br>O | | | | | |
| 5 | Lu(a+)<br>Le(a−b+)<br>O | Lu(a−)<br>Le(a+b−)<br>$A_1$ | Lu(a−)<br>Le(a+b−)<br>O | Lu(a−)<br>Le(a+b−)<br>$A_1$ | Lu(a−)<br>Le(a+b−)<br>$A_1$ | identical twins | | | |
| 6 | Lu(a−)<br>non-sec.<br>Le(a+b−)<br>L<br>■ | Lu(a+)<br>sec.<br>Le(a−b+)<br>L<br>$A_1$ | Lu(a+)<br>sec.<br>Le(a−)<br>L<br>$A_1B$ | Lu(a−)<br>sec.<br>Le(a−)<br>L<br>$A_1B$ | Lu(a+)<br>sec.<br>Le(a−b+)<br>L<br>O | | | | |
| 7 | Lu(a+)<br>Le(a−b+)<br>$A_1$ | Lu(a−)<br>Le(a−b+)<br>O | Lu(a−)<br>Le(a−b+)<br>$A_1$ | Lu(a+)<br>Le(a+b−)<br>O | | | | | |
| 8 | Lu(a−)<br>Le(a−b+)<br>$A_1$ | Lu(a+)<br>Le(a−b+)<br>$A_1$ | Lu(a−)<br>Le(a+b−)<br>$A_1$ | Lu(a−)<br>Le(a−b+)<br>$A_1$ | | | | | |
| 9 | Lu(a−)<br>Le(a−)<br>O | Lu(a+)*<br>Le(a−)<br>O | Lu(a+)<br>Le(a+)<br>O | Lu(a+)<br>Le(a−)<br>O | | | | | |
| 10 | Lu(a+)<br>Le(a−)<br>$A_1B$ | Lu(a−)<br>Le(a−)<br>$A_1$ | Lu(a−)<br>Le(a−)<br>$A_1B$ | Lu(a+)<br>Le(a+)<br>B | | | | | |
| 11 | Lu(a−)<br>sec.<br>Le(a−)<br>$A_1$ | Lu(a+)<br>sec.<br>Le(a−)<br>$A_1$ | Lu(a−)<br>sec.<br>Le(a−)<br>$A_1$ | Lu(a+)<br>non-sec.<br>Le(a+)<br>$A_1$ | Lu(a+)<br>Le(a−)<br>$A_1$ | Lu(a+)<br>sec.<br>Le(a−)<br>$A_1$ | | | |
| 12 | Lu(a−)<br>Le(a−)<br>$A_1$ | Lu(a+)<br>Le(a−)<br>B | Lu(a+)<br>Le(a−)<br>$A_1B$ | Lu(a−)<br>Le(a+)<br>$A_1$ | Lu(a+)<br>Le(a−)<br>$A_1B$ | | | | |
| 13 | Lu(a+)<br>sec.<br>Le(a−b+)<br>L<br>$A_1$ | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>$A_2$ | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>$A_2$ | Lu(a+)<br>sec.<br>Le(a−b+)<br>L<br>$A_1$ | Lu(a−)<br>non-sec.<br>Le(a−b−)<br>ll<br>$A_2$ | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>$A_1$ | Lu(a+)<br>sec.<br>Le(a−b−)<br>ll<br>$A_2$ | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>$A_1$ | |
| 14 | Lu(a+)<br>sec.<br>Le(a−b+)<br>L<br>O | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>O | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>O | Lu(a+)<br>sec.<br>Le(a−b+)<br>L<br>O | | | | | |
| 15 | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>O | Lu(a+)<br>sec.<br>Le(a−b+)<br>L<br>B | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>■ | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>B | | | | | |
| 16 | Lu(a−)<br>Le(a−b+)<br>B | Lu(a+)<br>Le(a−b+)<br>B | Lu(a−)<br>Le(a−b+)<br>B | Lu(a+)<br>Le(a−b+)<br>O | | | | | |
| 17 | Lu(a−)<br>Le(a−b+)<br>$A_1$ | Lu(a+)<br>Le(a−b+)<br>$A_1$ | Lu(a−)<br>Le(a−b+)<br>$A_1$ | Lu(a+)<br>Le(a−b+)<br>$A_1$ | | | | | |
| 18 | Lu(a−)<br>Le(a−b+)<br>O | Lu(a+)<br>Le(a−b+)<br>$A_1$ | Lu(a+)<br>Le(a−b+)<br>$A_1$ | Lu(a−)<br>Le(a−b+)<br>O | | | | | |
| 19 | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>$A_1$ | Lu(a+)<br>sec.<br>Le(a−b+)<br>L<br>O | Lu(a+)<br>Le(a−b−)<br>O | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>O | Lu(a−)<br>sec.<br>Le(a−b−)<br>ll<br>$A_1$ | | | | |
| ■ | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>O | Lu(a+)<br>sec.<br>Le(a−b+)<br>L<br>$A_1$ | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>O | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>$A_1$ | Lu(a+)<br>sec.<br>Le(a−b+)<br>L<br>O | Lu(a+)<br>Le(a−b+)<br>O | | | |

TABLE 2—*continued*

| No. | Father | Mother | Children | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | 1 | 2 | 3 | 4 | 5 | 6 | 7 |
| 21 | Lu(a−)<br>Le(a−b+)<br>$A_1$ | Lu(a+)<br>Le(a−b+)<br>$A_2B$ | Lu(a+)<br>Le(a−b+)<br>$A_1$ | Lu(a−)<br>Le(a−b+)<br>B | Lu(a−)<br>Le(a−b+)<br>$A_1B$ | | | | |
| 22 | Lu(a+)<br>Le(a−)<br>O | Lu(a−)<br>Le(a−)<br>O | Lu(a−)<br>Le(a−)<br>O | Lu(a+)<br>Le(a−)<br>O | | | | | |
| 23 | Lu(a−)<br>Le(a−)<br>O | Lu(a+)<br>Le(a−)<br>$A_1$ | Lu(a−)<br>Le(a−)<br>$A_1$ | Lu(a−)<br>Le(a−)<br>$A_1$ | | | | | |
| 24 | Lu(a−)<br>Le(a−)<br>$A_1$ | Lu(a+)<br>Le(a−)<br>O | Lu(a+)<br>Le(a−)<br>$A_2$ | Lu(a+)<br>Le(a−)<br>$A_2$ | | | | | |
| 25 | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>B | Lu(a+)<br>sec.<br>Le(a−b+)<br>L<br>O | Lu(a+)<br>Le(a−b+)<br>O | Lu(a+)<br>sec.<br>Le(a−b+)<br>L<br>B | | | | | |
| 26 | Lu(a+)<br>Le(a−b+)<br>O | Lu(a+)<br>Le(a−b+)<br>$A_1$ | Lu(a−)<br>Le(a−b+)<br>O | Lu(a+)<br>Le(a−b+)<br>$A_1$ | | | | | |
| 27 | Lu(a+)<br>Le(a−b+)<br>B | Lu(a+)<br>Le(a−b+)<br>O | Lu(a+)<br>Le(a−b+)<br>O | Lu(a+)<br>sec.<br>Le(a−b+)<br>L<br>B | | | | | |
| 28 | Lu(a+b+)<br>Le(a−b−)<br>O | Lu(a+b+)<br>Le(a−b+)<br>O | Lu(a+b+)<br>Le(a−b+)<br>O | Lu(a+)<br>O | Lu(a+b+)<br>Le(a−b+)<br>O | Lu(a+b−)<br>Le(a−b+)<br>O | | | |
| 29 | Lu(a+)<br>Le(a−b+)<br>O | Lu(a+)<br>Le(a−)<br>$A_1$ | Lu(a+)<br>Le(a−b+)<br>O | Lu(a+)<br>Le(a−b+)<br>O | | | | | |
| 30 | Lu(a−)<br>Le(a−b+)<br>B | Lu(a+)<br>Le(a+b−)<br>O | Lu(a+)<br>Le(a−b+)<br>B | Lu(a+)<br>Le(a+b−)<br>B | | | | | |
| 31 | Lu(a−)<br>Le(a−b+)<br>B | Lu(a+)<br>Le(a+b−)<br>B | Lu(a+)<br>Le(a+b−)<br>B | Lu(a+)<br>Le(a+b−)<br>B | | | | | |
| 32 | Lu(a−)<br>Le(a−b+)<br>$A_1$ | Lu(a+)<br>Le(a+b−)<br>O | Lu(a+)<br>Le(a−b+)<br>O | Lu(a+)<br>Le(a−b+)<br>O | | | | | |
| 33 | Lu(a−)<br>Le(a−b−)<br>O | Lu(a+)<br>Le(a+b−)<br>O | Lu(a+)<br>Le(a−b+)<br>O | Lu(a+)<br>Le(a−b+)<br>O | | | | | |
| 34 | Lu(a−)<br>Le(a−)<br>$A_1$ | Lu(a+)<br>Le(a+)<br>$A_2$ | Lu(a−)<br>Le(a+)<br>$A_1$ | Lu(a+)<br>Le(a+)<br>$A_1$ | Lu(a+)<br>Le(a−)<br>$A_2$ | Lu(a+)<br>Le(a−)<br>O | Lu(a−)<br>Le(a−)<br>$A_2$ | Lu(a−)<br>Le(a−)<br>O | dissimilar quadruplets |
| 35 | Lu(a−)<br>Le(a−)<br>$A_1$ | Lu(a+)<br>Le(a+b−)<br>O | Lu(a+)<br>Le(a−)<br>O | Lu(a−)<br>Le(a−)<br>O | | | | | |
| 36 | Lu(a+)<br>Le(a+)<br>B | Lu(a−)<br>Le(a−)<br>$A_1$ | Lu(a+)<br>Le(a−)<br>O | Lu(a−)<br>Le(a−)<br>B | Lu(a−)<br>Le(a−)<br>B | Lu(a+)<br>sec.<br>Le(a−)<br>L<br>$A_1B$ | | | |
| 37 | Lu(a−)<br>Le(a+b−)<br>O | | Lu(a+)<br>Le(a−b−)<br>O | Lu(a−)<br>Le(a−b+)<br>$A_1$ | dissimilar twins | | | | |
| 38 | Lu(a−)<br>sec.<br>Le(a−b−)<br>ll<br>O | | Lu(a+)<br>Le(a−b+)<br>O | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>$A_2$ | | | | | |
| 39 | Lu(a+)<br>$A_1$ | Lu(a+)<br>Le(a−b+)<br>AB | Lu(a−)<br>Le(a+b−)<br>AB | Lu(a+)<br>Le(a−b+)<br>$A_1B$ | Lu(a+)<br>Le(a−b+)<br>B | | | | |
| 40 | sec.<br>L | Lu(a+)<br>sec.<br>Le(a−b+)<br>L<br>O | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>O | Lu(a−)<br>sec.<br>Le(a−b+)<br>L<br>O | | | | | |

Some of the unscored families could be scored but the results would be negligible. Some of the families (*e.g.* 31-36) are probably unscorable but we thought it better to include them.

* The mother in family 9 is heterozygous $Lu^aLu^b$ because her mother is $Lu^bLu^b$

L = Le$^a$ substance present in the saliva : ll = Le$^a$ substance absent.

For more subtle analysis of the families shown in table 2 the following Caucasian gene frequencies will be needed :

| | | | |
|---|---|---|---|
| *Lu*$^a$ | 0·0390 | *Se* | 0·5077 | *L* | 0·6870 |
| *Lu*$^b$ | 0·9610 | *se* | 0·4923 | *l* | 0·3130 |

The *Lu*$^a$ and *Lu*$^b$ frequencies are taken from Race and Sanger (1958) ;

TABLE 3

*Linkage analysis of the "certain" families of table 2*

| Comparisons | Families scored | $\Sigma(\lambda)$ | $\Sigma(\kappa)$ | $2{\cdot}33\sqrt{\Sigma(\kappa)}$ | $\frac{1}{2}\left[1-\sqrt{\frac{\Sigma(\lambda)}{\Sigma(\kappa)}}\right]$ |
|---|---|---|---|---|---|
| Lutheran : secretor . | 1, 2, 3, 11, 13 | +17·172 | 34·660 | 13·7 | 0·15 |
| Lutheran : *Ll* . . | 13 and 19 | −1·387 | 2·232 | 3·5 | ... |
| Secretor : *Ll* . . | 13 | +0·057 | 1·059 | 2·4 | ... |
| Lutheran : Le$^a$ phenotype of red cells | 1, 2, 3, 4, 5, 7, 8, 9, 10, 11, 12 | +19·792 | 38·667 | 14·5 | 0·14 |

TABLE 4

*The linkage results of Mohr and of Ceppellini and Siniscalco added to our own*

| Comparison | Source | $\Sigma(\lambda)$ | $\Sigma(\kappa)$ | $2{\cdot}33\sqrt{\Sigma(\kappa)}$ | $\frac{1}{2}\left[1-\sqrt{\frac{\Sigma(\lambda)}{\Sigma(\kappa)}}\right]$ |
|---|---|---|---|---|---|
| Lutheran : secretor . | Present paper . . | +17·172 | 34·660 | 13·7 | 0·15 |
| Lutheran : *Ll* . . | Ceppellini and Siniscalco | −0·556 | 4·333 | | |
| | Present paper . . | −1·387 | 2·232 | | |
| | Total . . | −1·943 | 6·565 | 6·0 | |
| Secretor : *Ll* . . | Ceppellini and Siniscalco | −3·556 | 41·358 | | |
| | Present paper . . | +0·057 | 1·059 | | |
| | Total . . | −3·499 | 42·417 | 15·2 | |
| Lutheran : Le$^a$ phenotype of red cells . | Mohr . . . . | +48·965 | 62·556 | | |
| | Present paper . . | +19·792 | 38·667 | | |
| | Total . . | +68·757 | 101·223 | 23·4 | 0·09 |

the *Se* and *se* frequencies are taken from Clarke *et al.* (1956) ; the *L* and *l* frequencies are taken from Grubb (1951). The following approximate red cell phenotype frequencies may also be of use : Le(a+b−) 22 per cent., Le(a−b+) 72 per cent., Le(a−b−) 6 per cent.

## 3. DISCUSSION

Table 4 shows that the Lutheran and secretor genes are linked : it also shows that the Lutheran genes are linked to genes responsible for the Lewis phenotypes of the red cells. According to the present view the Lewis phenotypes of the red cells are controlled by the *Ll* genes and the secretor genes. The evidence against linkage between the *Ll* genes and the secretor genes is weighty. Therefore the linkage between the Lutheran genes and the Lewis phenotype of the red cells can most economically be explained by the Lutheran-secretor linkage : there is no need to postulate linkage between the Lutheran genes and the *Ll* genes and, indeed, the direct evidence against such linkage is slowly being collected. The collection is bound to be slow for the only really informative type of mating, $Lu^aLu^b$, $Ll \times Lu^bLu^b$, *ll* has a frequency in England of three in a thousand, and, to contribute usefully the mating would have to have produced, at the very least, four children.

At present then it seems certain that the Lutheran genes are linked to the secretor genes and most unlikely that they are linked to the Lewis genes as we now understand them.

## 4. SUMMARY

The first autosomal linkage to be recognised in Man was that discovered by Mohr between the Lutheran genes and genes responsible for the Le(a+) and Le(a−) reactions of red cells. The linkage gains support from our results which, when combined with those of Mohr, suggest a recombination fraction of 0·09.

It is now becoming clear that the linkage is in fact between the Lutheran genes and the secretor genes and not between the Lutheran and Lewis (*Ll*) genes. The appearance of linkage with the Lewis genes is due to their phenotypic expression on red cells being controlled by the secretor genes.

*Acknowledgments.*—We wish to thank Dr W. G. D. Murray, of Greenhithe, and Dr C. A. Holman, of the Lewisham Hospital, for the samples of blood and saliva from family No. 1. We are grateful to the family of Professor C. D. Darlington, F.R.S. (family No. 2) for giving their blood and for having such informative groups.

Past and present members of the Unit responsible for some of the family groupings are Dr Sylvia D. Lawler, Mrs Joan Sneath, Miss Helene Holt, Miss Phyllis Moores and Miss Jean Noades.

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# REVIEWS

**A CENTURY OF DARWIN.** By S. A. Barnett *et al.* London: Heinemann. 1958. Pp. xvi+376. 30s.

**A HANDBOOK OF EVOLUTION.** British Museum (Natural History). London. 1958. Pp. ix+110. 5s.

A number of publications have appeared to celebrate the centenary of the Darwin-Wallace paper. These are two of very different kinds.

*A Century of Darwin* is a symposium by fifteen contributors which cover not unsuccessfully, but in a random order, the wide range of Darwinian studies. In their methods they also cover a wide range. Six of them are devoted to specific themes of Darwin : the Descent of Man, Sexual Selection, Animal Breeding, Expression of Emotions, Coral Reefs, and Palæontology are all most instructive. Heslop Harrison's account of the six plant books in which Darwin appears as an experimenter and a founder of population genetics is also highly topical. Articles by Le Gros Clark on Man and by de Beer on Development are particularly well illustrated.

These are aspects of Darwin's work about which there is least controversy. More disputable are his historical and sociological connections. We must expect a few injustices to Darwin's predecessors. It was Lamarck not T. H. Huxley who invented the word and partly the idea of biology (p. 122). Erasmus Darwin's greatest work on animals, Zoonomia, was in prose not verse (p. 1). Nor were his other works " long and unfortunately remarkably boring poems ". The size of his *Temple of Nature* was due to its copious and closely reasoned scientific notes. Charles Darwin misrepresented these works without reading them, T. H. Huxley followed his example, and the practice has held for a century.

MacRae, writing on Social Sciences, supplies another dubious connection for he considers that Darwinism derived from Malthus. He does not consider that both might be derived from Erasmus Darwin. MacRae, turning from history to our present state, concludes that the social sciences need something other than, or more than " Darwinism ". The last word in biology, he writes, is no longer the first word in sociology. But what is the last word in biology ? There is no evidence that this writer knows it or is looking for it.

Four articles discuss recent genetic work. Dobzhansky is rich in recent research on species. Thoday considers the genetic properties of breeding groups in relation to natural selection. Waddington describes *Drosophila* experiments in which he found that selection under one set of conditions produced a genetic change which had a correlated effect under another, more normal, set of conditions. He does not, however, make it clear what other result might have been expected. Nor is it clear to me what bearing this result has on selection theory.

The most exciting of the articles is that entitled " The Third Stage of Genetics " by Dr Donald Michie of the Royal Veterinary College. Here important experiments are given a new or at least a newly connected interpretation. Briggs and King have shown by transplantation that nuclei change in the course of development in the frog. By transplantation also

Danielli has shown that nuclei change when they are put in a foreign cytoplasm. Dr Michie seems to think that these changes mean a modification of heredity. But does this follow? Could it not be the nucleoli which change? Is it not supposed that nuclear genes by interaction with the cytoplasm produce what some people call " plasmagenes " concerned with differentiation? And (on the lines Caspersson suggested in 1941) might these not be housed in the nucleolus on the way out?

Turning to experiments with plants, Michie quotes the Rye-Wheat example of the induced diffusion of a self-propagating particle. This consequence of grafting has been inferred in various plants over a period of thirty years (Hoffmann, F. W., 1927, *J. Agric. Res.*, *34*, 673). So frequently indeed that I have used the expression *provirus* to cover the situation. The term has even been extended to apply to the somewhat different relation between stable or attached phage and the bacterium, a connection which Michie also seems to recognise.

So far in this third stage of genetics Dr Michie has been dealing with experiments which have been generally confirmed or supported by published work. They are text-book experiments. With these, however, he associates (so far as I understand his argument) two series of works which have not been confirmed. There is an experiment by Glavinic in Belgrade which is used to infer that grafted tomatoes will give progenies showing Mendelian ratios modified by the stock. It is a mistake, however, not to mention that attempts to repeat the earlier work on these lines of Glushchenko have failed, *e.g.* by Leo Sachs (1951, *Nature*, *167*, 282-3), and by Hans Stubbe (1954, *Kulturpflanze*, *2*, 185-236).

A similar mistake is made in quoting Benoit and others on the effects of DNA on ducks which is said to have " fired experimental biologists throughout the world " (p. 84). Perhaps, too, Dr Michie might have added to his discussion of Daniel's graft-hybrids references to the contrary opinions of workers who had experience of this kind of experiment. In this way the beginner, unfamiliar with the published work on his subject, may avoid being " fired " unnecessarily.

With these few words of caution (which will not be needed by most readers) *A Century of Darwin* may be warmly recommended.

*Evolution* is published as a handbook to the centenary exhibition, organised by Sir Gavin de Beer, which has this year transformed the central hall of the British Museum of Natural History. It is a popular and well illustrated account of the history of Darwinism, of the present views of evolution showing in detail the impact of recent genetic theory and experiment on them. It should be valuable for schools and the general reader. There is a list of references. C. D. D.

NATURAL SELECTION AND HEREDITY. By P. M. Sheppard. London: Hutchinson. 1958. Pp. 212. 18s.

Natural Selection and Heredity together constitute a formidable subject. Dr Sheppard, in this relatively small book, tackles it by selecting a number of topics to illustrate the important principles. He begins with an admirable chapter on the history of the concept of natural selection in biology. There follow chapters on elementary genetics, including polygenic inheritance, on the evolution of dominance, mutation and genetic drift. A large section,

including most of the examples, is devoted to polymorphism, with reference to mimicry and protective coloration mainly in moths and butterflies. The book ends with ecological genetics, the origin of species and a concluding chapter of a general nature on evolution.

These selected topics are well, often very well, treated. The writing generally is direct and clear, and the examples of experiments and observations well chosen. Considered as a whole, however, the book is disappointing, The " important principles ", one feels, would have emerged with greater force if, before dealing with selection, the author had clearly defined the nature of the genetic systems within, and upon, which natural selection operates: also, how the variables within the systems—the reproductive mechanisms, breeding behaviour, etc.—are interrelated. It would then have been easier for the reader to grasp more fully the implications of change at various levels within the genetic framework of a population. As it is the book is a valuable record of Natural Selection at work, especially for advanced University students. In surveying modern views on the theory of natural selection, however, it is much less satisfactory.

H. Rees.

**GENETICS AND THE IMPROVEMENT OF TROPICAL CROPS. By Sir Joseph Hutchinson. Cambridge University Press. 1958. Pp. 1-30. 3s. 6d.**

This is the inaugural address of the Draper's Professor of Agriculture in the University of Cambridge. Its main theme centres on the principles underlying the distribution and utilisation of genetic variation in cultivated plants. Vavilov's general conclusion on the reduction in variability away from the centre of origin is challenged, and a distinction based on the breeding system is made between species. It is suggested that the Vavilov effect may be the result of loss of recombination brought about by enforced inbreeding in peripheral regions. In *Gossypium hirsutum* no variation which could be used in a breeding programme was revealed in extensive collections made at the centre of origin, while newer centres of diversity in Africa and Asia have provided valuable material for the improvement of the cotton crop. Great stress is laid on the importance of defining precisely the objectives of a breeding programme and an analysis is made of the outstandingly successful crop improvement projects in parts of Africa. It is a thoughtful and stimulating essay by one who is the undoubted authority on the subject.

Watkin Williams.

# GENETICAL SOCIETY OF GREAT BRITAIN

## ABSTRACTS of Papers read at the HUNDRED AND TWENTY-SEVENTH MEETING of the Society, held on 28th JUNE 1958, at UNIVERSITY COLLEGE, LONDON

## THE INFLUENCE OF THE ENVIRONMENT IN ANENCEPHALY

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Data on 4420 anencephalics born in Scotland between 1939 and 1956 are presented, various environmental features associated with differing incidence are discussed, and some limitations of the nature-nurture issue in relation to observational data are considered.

The basic findings are :—(1) There is a marked association with social class, the variation in incidence being fourfold. (2) There is a consistently lower incidence in illegitimate pregnancies. (3) The liability in first pregnancies is 40 per cent. higher than in second. (4) The incidence probably increases with maternal age, but may also be relatively high under the age of 20. (5) There is a marked regional variation, but urbanisation itself does not seem to be a factor. (6) The marked secular changes are largely related to a changing incidence in first births and in winter births. (7) There is a marked seasonal variation, the incidence being highest in mid-winter. (8) There is evidence that epidemic infections are not related to any considerable number of cases.

## ACROCEPHALOSYNDACTYLY

C. E. BLANK

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Acrocephalosyndactyly is a rare malformation which may conveniently be divided into two main sub-groups, (i) Apert's syndrome and (ii) other types. This paper is mainly concerned with (i) which is the commonest type.

The deformities in Apert's syndrome have been attributed to syphilis, rickets, chronic osteitis, hormonal dysequilibrium and even to maternal rubella. Recent authors have considered a hereditary origin probable.

Analysis of the pedigrees of 34 sporadic cases in the author's British series makes it very likely that sporadic cases of Apert's syndrome are due to new mutation. The discovery in Holland of an affected mother and child confirms this suspicion.

Of great interest to the geneticist is the marked effect of parental age. An increase in the mean ages of the parents at births of propositi can be attributed wholly to a raised paternal age ; so also can the associated shift in the birth order. In this respect the condition is very like achondroplasia.

Estimates of the incidence of Apert's syndrome at birth and in the general population are given.

## PHAGE-MEDIATED TRANSDUCTION OF COLICINOGENY IN *SALMONELLA TYPHIMURIUM*

H. OZEKI and B. A. D. STOCKER

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Colicinogeny, *i.e.* ability to produce a colicine (an antibiotic active on certain coliform bacteria), is transferred with high frequency to cells of a non-colicinogenic

strain when these are mixed with cells of a suitable colicinogenic strain, the transfer probably resulting from cell conjugation. *Shigella sonnei* strain P9 produces two different colicines, which we term $C_1$ (probably colicine I) and $C_2$ (probably colicine E). When a non-colicinogenic *Salmonella typhimurium* strain ($LT_2$) was mixed with P9, two different kinds of colicinogenic *Salmonella* were obtained, some clones producing colicine $C_1$ alone, others both $C_1$ and $C_2$; no *Salmonella* producing colicine $C_2$ alone have been detected in these experiments. Similar results were obtained when a doubly colicinogenic *Salmonella* strain thus produced was mixed with a (genetically marked) non-colicinogenic *Salmonella* strain.

In transduction experiments phage $PLT_{22}$ grown on the doubly colicinogenic *Salmonella* was applied to a non-colicinogenic *Salmonella* strain; some colicinogenic clones were obtained, their frequency being about the same as in the transduction of other characters. Of the clones made colicinogenic by transduction all those so far tested produce only colicine $C_2$, a class not encountered at all when colicinogeny was transferred between the same strains by mixing the cultures.

## COMBINATION OF INFORMATION FROM 2×2 TABLES

B. WOOLF
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Provided attention is confined to only two blood types, the data for studying the relation between blood group and disease can be set out as a 2×2 table thus :—

| | TRAIT :—Blood Group | | |
|---|---|---|---|
| | *α* | *β* | |
| GENUS I : Patients | a | b | E |
| GENUS II : Control Series | c | d | F |
| | S | T | N |

Samples from two sub-populations or *genera* are sub-divided according to some *trait*. Data conforming to this pattern are common in genetics. Often there are several tables, the results of comparisons under different conditions such that the incidence of the trait would be expected to vary in both genera, but in such a way that there should be a more or less constant functional relation between the relative incidence figures in the different tables. Methods will be described for statistical analysis of such sets of data.

For the case of blood group and disease, Woolf (1955) suggested the use of the *relative incidence ratio*, $x = ad/bc$. For a set of tables, a weighted mean is computed of $y = \ln x$, with test of heterogeneity and significance. This *Empirical Estimate* is a " large sample " method, and breaks down entirely if any cell frequencies are zero. Simple computations will be described for obtaining the *Maximum Likelihood Estimate*, and for a rapid *Null Test and Estimate* which is often all that is needed.

These methods are valid for a wide class of postulated functional relationships, including for example comparative mutation rates. By use of the Null Test, the applicability of a selection of different " models " can be quickly assessed.

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# β-GLOBULIN POLYMORPHISM IN CATTLE AND OTHER MAMMALS

J. C. ASHTON

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Six β-globulin phenotypes have been found in the serum proteins of British breeds of cattle by starch gel electrophoresis. The six phenotypes are individual genotypes, being the homozygotes and heterozygotes of three alleles, $\beta^A$, $\beta^D$, and $\beta^E$. The gene frequencies differ markedly between breeds, $\beta^E$ being least frequent in each breed.

Abnormal segregation ratios have been obtained with certain matings. Thus $\beta^A/\beta^A \times \beta^A/\beta^D$ and $\beta^D/\beta^D \times \beta^A/\beta^D$ result in a significant excess of offspring of the same genotype as the mother. The matings $\beta^A/\beta^E \times \beta^A/\beta^A$ and $\beta^D/\beta^E \times \beta^D/\beta^D$ result in a significant lack of offspring with $\beta^E$ in their genotype.

$\beta^E$ is clearly at a disadvantage, which may be partially offset by the apparent economic advantages of $\beta^E$ heterozygotes. There is evidence of a cline in the frequency of $\beta^E$.

β-globulin polymorphism has been found in sheep and goats. The genetic mechanism is analagous to that in cattle. Two β-globulin alleles are postulated in goats and five in sheep.

Serum protein polymorphism has also been detected in the thread-proteins of cattle, in the slow-alpha proteins of cattle, in the pre-albumins of horses, and in the serum proteins of white mice.

# INDEX


ABH substance, 513
*Acacia*, 199-212
accentrics, 5 *sqq.*
acrocephalosyndactyly, 525
adaptation, 169, 252, 253
*Æthionema*, 239, 246
affinity, 459 *sqq.*
ageing, 1 *sqq.*
*Agilops*, 301 *sqq.*
albuminiferous glands, 225
*Allium*, 3, 196
allotetraploid, 240
*Alopecurus*, 122
amnion, 215
*Amphidasys*, 51
amphidiploids, 301 *sqq.*
anatomy, comparative, 271
anencephaly, 525
aneuploids, wheat, 396
aneuploidy, 228
animal breeding, 499
anthers, abnormal, 346
antigen-antibody system, 233, 244
antigens, 141
*Antirrhinum*, 24, 236, 243, 245
apomixis, 247
ascospore, 256
ascus, 373 sqq.
Ashton, J. C., 527
*Aspergillus*, 326, 329, 393
assymetry, sternopleural, 402 *sqq.*
autotetraploid, 116, 250
Avdulov, 441

backcross, 89, 199, 240
back-mutation, 262, 265
balance, 195, 201 *sqq.*
balanced lethal, 247 *sqq.*
Barber, H. N., 1-25
barley, 494
Barnett, S. A., 398, 521
Bateman, A. J., 213-232, 467-475
Bateman, N., 394
Bates, M., 139
behaviour, 463
Benoit, 522
biotic potential, 211
*Biston*, 51 *sqq.*
Blank, C. E., 525
blood group, ABO
  Lewis, 513, 514
  Lutheran, 513
blood type, sheep, 142
Bodmer, W. S., 363
body size, 213
Bradshaw, A. D., 143
*Brassica*, 240, 417 *sqq.*
breeding season, 349
Brian, P. W., 143
Briggs, F. N., 521
Brix, K., 441-452
buffering, 412
*Bythoscopus*, 210

*Campanula*, 130, 252
canalisation, 401, 404, 413
cancer, 268
Cannon, H. Graham, 271
*Capsella*, 239, 242
*Capsicum*, 234, 243
Carter, G. S., 271
Caspersson, T., 522
Cattenach, B. M., 141
cell, abnormal division, 444
  multinucleate, 422 *sqq.*
  size, 441
centromere, 2, 12, 90, 91, 141, 458
  daughter, 115
  subterminal, 114
centrotype, 459
certation, 340 *sqq.*
chaeta number, 401
Chapman, V., 301
chiasma, 101 *sqq.*, 429 *sqq.*, 445
  formation, 101, 459
  frequency, 101, 111, 122, 201, 457, 459
chimaera, 200
chlorosis, 200, 209
chorion, 217
chromatids, 115
chromatographic methods, 144
chromosome, acentric, 467
  acrocentric, 141
  addition, 301, 396
  arms, 90
  association, 445, 446
  behaviour, 101 *sqq.*, 127 *sqq.*
  breakage, 1 *sqq.*, 114, 115, 213 *sqq.*, 468, 495
  bridge, 229
  chain, 189
  coorientation, 445, 446
  deficiency, 250, 468
  deletion, 248
  disjunction, 189, 201
    frequency, 127, 129, 130
  duplication, 250, 467
  helix, 13
  inversion, 480
    bridge, 445, 446
  lagging, 443, 447, 450
  mapping, 91 *sqq.*, 429
  metacentric, 39, 141
  morphology, 309

chromosome, nucleolar, 127
number, 141, 201, 441, 443
pairing, 189, 201
polymorphism, 141
rearrangement, 213
ring, 189 *sqq.*, 238
sticky, 445, 449, 450
supernumerary X, 196
telocentric, 468
thread, 114
trabant, 10
X-, 189
cistron, 329
Clark, J. Le Gros, 521
Clausen, R. E., 301
cleavage abnormalities, 214
Clelland, 251
clonal variation, 444
clone, 441
cochlea, 465
Coleman, E. M., 398
colicinogeny, 525
competition, 154
complementary genotypes, 79, 85
component of variation, additive, 477 *sqq.*
non-additive, 477 *sqq.*
contagion factor, 21-23
Cooper, J. P., 400
Copp, L. G., 385, 390
corpus luteum, 214 *sqq.*
*Cosmos*, 242
*Crepis*, 242
crop improvement, 393
Crosby, J. L., 399
crossability, interspecific, 337
cross fertilisation, 117, 122
cross pollination, 113, 119, 121, 341, 343, 349
Crowe, L. K., 233-256
cytochemistry, 137
cytochrome deficiency, 258
cytology, 113
cytoplasm, 1, 493 *sqq.*
cytoplasmic, inheritance, 345, 354
variation, 393

Daday, H., 169-184
Danielli, 522
Darwin, C., 139, 271, 521
Darwin, E., 521
*Datura*, 342
Davies, D. R., 393
deafness, mouse, 463 *sqq.*
de Beer, Sir G., 521, 522
deciduoma, 214 *sqq.*, 467 *sqq.*
dehydrogenase, 234 *sqq.*
Deiter's cells, 465
deletions, 13
Deol, M. S., 463-476
diakinesis, 114
diallel cross, 102, 381 *sqq.*, 477 *sqq.*
differentiation fluids, 405
diffusion process, 43
disjunction, 127
disruptive selection, 398
distribution, binomial, 217
Eulerian, 20
gene frequency, 142
geographical, 144
negative binomial, 18, 19, 20
Poisson, 2, 18, 19, 469
Polya, 22
rectangular, 8
DNA, 138
labelled, 227
Dobzhansky, Th., 37-49, 521
dominance, 242, 408, 478
effects, 371 *sqq.*
interaction, 73 *sqq.*
dominant lethal, 467 *sqq.*
dosage time, 1
Dowrick, G. J., 393
*Draba*, 239
*Drosophila*, 1, 27 *sqq.*, 111, 196, 213, 230, 231, 250, 361, 401 *sqq.*, 458, 478, 496
species, 37 *sqq.*
dwarfism, genetic, 143

ecology, 253
*Ectropis*, 60
Edwards, J. H., 525
Edwards, R. G., 141
egg, mouse, 467 *sqq.*
production, 285 *sqq.*
Elliott, C. G., 398, 429-439
embryo sac, 247
embryonic death, 407 *sqq.*
endosperm, 480
*Endymion*, 431, 436
environmental, deterioration, 145
variation, 217
enzyme, activity, 321
extraction, 317
Ephrussi, B., 258, 266
*Epilobium*, 238, 248
epistacy, 371 *sqq.*, 478
*Eriophila*, 239
*Erysiphe*, 307
*Eucalyptus*, 177
*Euchlaena*, 495
*Eustreptocarpus*, 333
Evans, J. V., 142
evolution, 136, 145, 233, 252, 271
excretion, ethanolamine phosphate, 395
expressivity, 427

Falconer, D. S., 456
fermentation, 373
fertility, 123, 188, 200, 285 *sqq.*, 337
fertilisation rate, 142, 214 *sqq.*
*Festuca*, 449
Fincham, J. R. S., 317
Finney, D. J., 399
Fisher, Sir R. A., 133-135, 370
fitness, 253
internal, 153
sex difference, 155

flowering date, *Nicotiana*, 490
fœtal death, 214 *sqq.*
  resorption, 214
Ford, C. E., 141
*Fragaria*, 495
Fyfe, V. C., 370

Gale, J. S., 97-100
gametic elimination, 208 *sqq.*
gametophytic homomorphy, 233 *sqq.*
gamete formation, 358
gene, action, 480
  additive, 371 *sqq.*, 412
  autosomal, 97 *sqq.*
  complexes, unbalanced, 413
  conversion, 273, 283, 319
  dominant, 418
  enzyme relationships, 317
  flow, 51, 395
  frequency, 169 *sqq.*
    cline, 169
  inhibitor, 427
  interaction, 282, 414
  modifying, 64, 427
  multiple, 282
  stability, 412
general combining ability, 477, 483
generation, effect, 373
  matrix, 97 *sqq.*
  time, 146 *sqq.*
genetic, instability, 257
  value, 499
genotype, complementary, 79, 85
  environment interaction, 412
  ordered, 146 *sqq.*
germ cells, mouse, 213 *sqq.*
germination, 7
  date, *Primula*, 399
gestation length, 214
Gilbert, N. E. G., 477-492
Gildenhuys, P., 441-452
Gilmour, D. G., 141
Glavinic, 522
Glushenko, 522
glutamic dehydrogenase, 317 *sqq.*
*Godetia*, 234, 248
Goodwins, I. R., 73-75
Goossens, A. P., 441
graft hybrid, 522
growth, twin cattle, 396

Hamerton, J. L., 141
Hamilton, H., 391
haploid, 3
haplont, 154
Harland, S. C., 27-36
Harris, H., 142, 144, 395
hatchability, 285 *sqq.*
Hawthorne, D. C., 273
Hayman, B. I., 371
*Haynaldia*, 302
heritability, 413, 414
hermaphroditism, 143
Heslop-Harrison, J., 143
heteroalleles, 330
heterocaryon, 320 *sqq.*
  complementarily, 326
  forced, 322, 324
heterochromatin, 2, 3, 12, 189, 344, 482, 486
heterogeneity, genetic, 401
  tests, 359
heterosis, 34, 36 *sqq.*, 343, 344, 381 *sqq.*, 482, 486
  index, 344
  mice, 398
  single gene, 397
heterostyly, 233
heterozygosity, 401
heterozygote, double, 127, 128
  interchange, 127, 128, 185 *sqq.*
  inversion, 37 *sqq.*, 46, 115
Hiraizumi, T., 154
Hoffmann, F. W., 522
homeostasis, 47, 312, 401 *sqq.*
homostyle, 363
homozygosity, 401, 411
*Hordeum*, 241, 493 *sqq.*
Hubbard, 441
Humphrey, P. S., 139
Hutchinson, Sir J., 523
Huxley, T. H., 521
*Hyacinthus*, 433
hybrid, structural, 185
hybridity, optimum, 193
*Hymencallis*, 449
*Hyoscyamus*, 348
*Hypericum*, 252
hypophosphatasia, 395

*Iberis*, 239
illegitimacy, 369
implantation, mouse, 214, 467
  reaction, 214, 215
inbred lines, 101
inbreeding, 107, 141, 146, 153, 185, 195, 253, 401, 454, 459, 480, 481, 505
  coefficient, 42, 286, 288
  depression, 33, 124
  double cousin, 97 *sqq.*
  intra-generation, 285
  uncle-niece, 97 *sqq.*
incompatibility, self, 118, 123, 233 *sqq.*
  unilateral interspecific, 233 *sqq.*
interaction, genetical, 477, 478
  genotype-environment, 412
  statistical, 477, 482 *sqq.*
intercept length, 90, 361
interference, 78, 89 *sqq.*, 357 *sqq.*
  function, 91
interspecific cross, 199
inversion, heterozygous, 46
  polymorphism, 46, 47
irradiation, 1 *sqq.*, 213 *sqq.*, 469 *sqq.*
  dose dependence, 474

Jackson, A. R. H., 27-36

Jackson, W. D., 1-25
Jamieson, A., 395
Jardine, R., 499
J character, 395
John, B., 185-197

Kalmus, H., 391
kappa factor, 496
karyotype, 38, 39, 47
Kettlewell, H. B. D., 51-72
Killick, D., 441
Kimura, M., 145-167
King, J. W. B., 142
Kitchin, F. D., 144
Kocher, W., 463-466
Kosambi coefficient, 89, 360, 361
*Kotochalia*, 210

Lamark, 271, 521
*Lathyrus*, 122
latin square, 357, 360, 486
Lawrence, C. W., 127-131
Lawrence, W. J. C., 333
*Layia*, 242
leaf, area, 344
  characters, *Acacia*, 199 *sqq.*
*Lepidium*, 239
lethal, 199, 469
  dominant, 213 *sqq.*
  imitation, 247
  mechanical, 469
  recessive, 213
Levene, H., 37-49
Lewis, D., 233-256
Lewis, K. R., 185-197
Lima bean, 490
linkage, 77 *sqq.*, 111, 134, 146, 163, 195, 196, 207, 381
  group V, mouse, 357
  group XII, mouse, 453 *sqq.*
  Lutheran-secretor, 513
  quasi-, 459, 460
*Linum*, 240
litter size, 213, 214, 467
locus, homologous, 427
*Locusta*, 196
*Lolium*, 449
*Lycium*, 239, 245
*Lycopersicum*, 234, 243, *sqq.*
*Lygidolon*, 210

Macer, R. C. F., 307
MacRae, 521
Malthus, T. R., 521
maltose, 278
Markoff chains, 43, 44
matings, brother-sister, 141
*Matthiola*, 417
maximum likelihood, 481
McBride, G., 395
McLaren, A., 394
melanism, industrial, 51 *sqq.*
meiosis, 190 *sqq.*, 201, 227, 430, 444 *sqq.*
  abnormal, 449, 450
merogon, 497
Michie, D., 394, 521, 522
micronucleus, 443, 449, 467, 468
migration, 30
missing plot technique, 480
mitosis, 3, 114, 189, 441 *sqq.*
  abnormal, 444, 448, 449
  C-, 10
Moffett, A. A., 199-212
Moncrieffe, I., 139
Monte Carlo methods, 45
morphology, yeast colony, 257-270
mouse, 213-232, 394, 453 *sqq.*, 463 *sqq.*, 467 *sqq.*
  head tossing, 463
  mutants, 99, 453
Mowl, M., 370
multiple alleleomorphs, 241, 253, 479
mustard gas, 1, 3
mutagen, 1, 213, 258
mutagenic sensitivity, 213-232
mutation, 1, 213 *sqq.*, 233 *sqq.*, 259 *sqq.*, 261, 328, 355, 414, 427
  in crop plants, 393
  induced, 393
  rate, 60 *sqq.*, 472 *sqq.*
muton, 329

natural selection, 64, 145-167, 442, 522
*Neurospora*, 1, 317
*Nicotiana*, 209, 245, 301, 377 *sqq.*, 490, 491, 495
Nixon, K., 199-212
non-disjunction, 189
non-reduction, 247
nucleic acids, 137, 138

*Œnothera*, 127, 130, 195, 233 *sqq.*
Oparin, A. I., 136
organ of Corti, 465
origin of life, 136
otolith, 465
outbreeding, 459, 460
  mechanisms, 252
outcrossing, 77, 366, 369
ovary, 417
overpopulation, 145, 164
ovulation, 213
Ozeki, H., 525

*Pachys*, 51
*Paonia*, 194
palæontology, 271
Palmer, L. S., 139
Palmer, T. P., 417-428
*Papaver*, 245
*Paramecium*, 1, 496
parameter, Mathusian, 145-147
Parsons, P. A., 77 *sq.*, 357
parthenogenesis, male, 493
Pateman, J. A., 317
penetrance, 423, 428
*Pennisetum*, 441 *sqq.*

Penrose, L. S., 144
*Periplaneta*, 185-197
*Petunia*, 233, 244
*Phalaris*, 241
*Phleum*, 241
*Phœnicorus*, 52
photosynthesis, 137
*Pisum*, 177, 252
placenta, 214, 215
plant height, *Nicotiana*, 491
pleiotropy, 111, 207
polarisation, 2, 12
pollen, germination, 238
  grain size, 341, 342, 355, 447
  mother cell, 229, 433, 444, 445
  tube growth, 235 *sqq.*
pollination, natural, 336
polygenic inheritance, 367
polymer formation, 136
polymorphism, 46, 55, 65, 141
  β-globulin, 527
  balanced, 64 *sqq.*, 70
  chromosomal, 37, 40
polyploidy, 441, 450
population, effective size, 42
  fitness, 145-167
  size, 42
  variability, 160
position effect, 248
potence, 484
Pottinger, D., 139
poultry, 141, 285, 509
*Primula*, 241, 363-370
production index, 287 *sqq.*
progeny test, 509
prototrophs, yeast, 257, 270
*Prunus*, 246, 251
pseudopregnancy, 213 *sqq.*
pseudowilds, 318
*Puccinia spp.*, 307

Race, R. R., 513
radiation, 1, 139
random mating, 149, 151
  population, 481
*Raphanus*, 240, 417
reciprocal, cross, 234 *sqq.*, 423, 477
  difference, 234
  interchange, 248
recombination, 46, 77, 83, 88 *sqq.*, 107, 414, 429, 468
  *Aspergillus*, 398
  double, 90
  estimation, 360, 361
  fraction, 87, 163
  super, 93
  value, 88, 453 *sqq.*
recon, 329
Rees, H., 101 *sqq.*, 522, 523
Reeve, E. C. R., 397
Reisig, J. L., 317, 397
*Rhœo*, 130, 195, 252
*Ricinus*, 177
Riley, R., 301, 396
Robson, E. B., 144, 395
Rolph, C. H., 139
Roper, J. A., 393
roundworm, sheep, 397
Rowlands, D. G., 113 *sqq.*
rye, 101 *sqq.*, 127 *sqq.*, 195
ryegrass, 400

*Saccharomyces*, 257-270, 273
sacculus, 465
Sachs, L., 522
saliva, 513 *sqq.*
*Salmonella*, 525
Sanger, R., 513
scaling, 406
*Secale*, 122, 241, 303 *sqq.*
seed setting, 337
seed size, lima bean, 490
segmental functions, 361
segregation, 141, 479
  artificial, 146, 399
selection, 127 *sqq.*, 161 *sqq.*, 257 *sqq.*, 401 *sqq.*, 459, 481
  coefficient, 145
  for heterozygosity, 185 *sqq.*
  for lactation, 394
  natural, 142, 143
  pressure, 63
  stabilising, 402
selective, advance, 510
  advantage, 71
  potential, 177
  value, 146
self, duplication, 137
  fertility, 156
  fertilisation, 119
  incompatibility, 118
  pollination, 107, 119, 127, 341, 343, 349
selfing, 124, 199, 418
*Senecio*, 242
sex difference, 77
shattering, grain, 385
Shepherd, P. M., 522
Sheppherd's correction, 368
sib tests, 509
significance, tests, 133
*Silene*, 143
skeletal morphology, 394
Snell, G. D., 459
*Solanum*, 234
somatic mutation, 268
species, cross pollinating, 113
  outbreeding, 108
speciation, *Streptocarpus*, 352
specific combining ability, 477
spermatogenesis, 141, 213
sperm storage, 225
spindle abnormalities, 448 *sqq.*
sporophytic homomorphy, 233 *sqq.*
Srb, A. M., 257-270
stability, 435
  developmental, 412

stability, phenotypic, 143
Stephenson, E., 390
sterility, 141
  male, 143
  semi-, 195, 209, 467
stochastic indeterminacy, 156
Stocker, B. A. D., 525
*Streptocarpus*, 333 *sqq.*, 352 *sqq.*
Stubbe, H., 522
supergene, 354, 355
superovulation, spontaneous, 217

Taylor, St C. S., 396
Tebb, G., 285
tectorial membrane, 465
temperature effect, winter, 169 *sqq.*
terminalisation, 195
Thoday, J. M., 313, 321, 398, 401 *sqq.*
Thompson, J. B., 101 *sqq.*, 399
tomato, 377 *sqq.*, 478, 491, 492
*Tradescantia*, 2, 252
transition function, 21
translocation, 141-144, 467
  heterozygosity, 217
*Trifolium*, 246 *sqq.*
  population, 169 *sqq.*
*Trillium*, 3
*Triticum*, 2, 303 *sqq.*
*Triton*, 497
*Tulipa*, 3

univalent, 189
Unrau, J., 396
Urey, 136

vaginal plug, 225
variation, additive, 146 *sqq.*
  dominance, 145 *sqq.*
  environmental, 201, 429 *sqq.*
  epistatic, 145 *sqq.*
  genetic, 145 *sqq.*, 432
  internuclear, 433
  interspecific, 202
  phenotypic, 143
  population, 146
Vavilov, N. I., 523
viability, 79, 82
*Vicia*, 11, 113 *sqq.*

Wallace, M. E., 453 *sqq.*
Warren, F. L., 142
Williams, G. J., 393
Williams, W., 523
wheat, 377
Wheaton-Smith, C., 392
Whitehouse, R. N. H., 393, 399
Woolf, B., 526
Wright, S., 154

X-ray sensitivity, 1

yield, maize, 489, 490
  tomato, 491, 492
  wheat, 399

*Zea*, 252, 449, 478, 489, 490
zygotic elimination, 208 *sqq.*

# HEREDITY — VOLUME ELEVEN

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## CONTENTS


PAGE

THODAY, J. M. (Sheffield). Homeostasis in a selection experiment . . . . . 401

PALMER, T. P. (New Zealand). Split petals in crucifers 417

ELLIOTT, C. G. (Glasgow). Environmental effects on the distribution of chiasmata among nuclei and bivalents and correlation between bivalents . . 429

GILDENHUYS, P., and BRIX, K. (Natal). Cytological abnormalities in *Pennisetum dubium* . . . . 441

WALLACE, MARGARET E. (Cambridge). New linkage and independence data for ruby and jerker in the mouse . . . . . 453

DEOL, M. S., and KOCHER, W. (London). A new gene for deafness in the mouse . . . . . 463

BATEMAN, A. J. (Manchester). The partition of dominant lethals in the mouse between unimplanted eggs and deciduomata . . . . . 467

GILBERT, N. E. G. (Bayfordbury). Diallel cross in plant breeding . . . . . 477

DAVIES, D. R. (Aberystwyth and Wantage). Male parthenogenesis in barley . . . . . 493

JARDINE, R. (Melbourne). Animal breeding and the estimation of genetic value . . . . . 499

SANGER, RUTH, and RACE, R. R. (London). The Lutheran-secretor linkage in man: support for Mohr's findings . . . . . 513

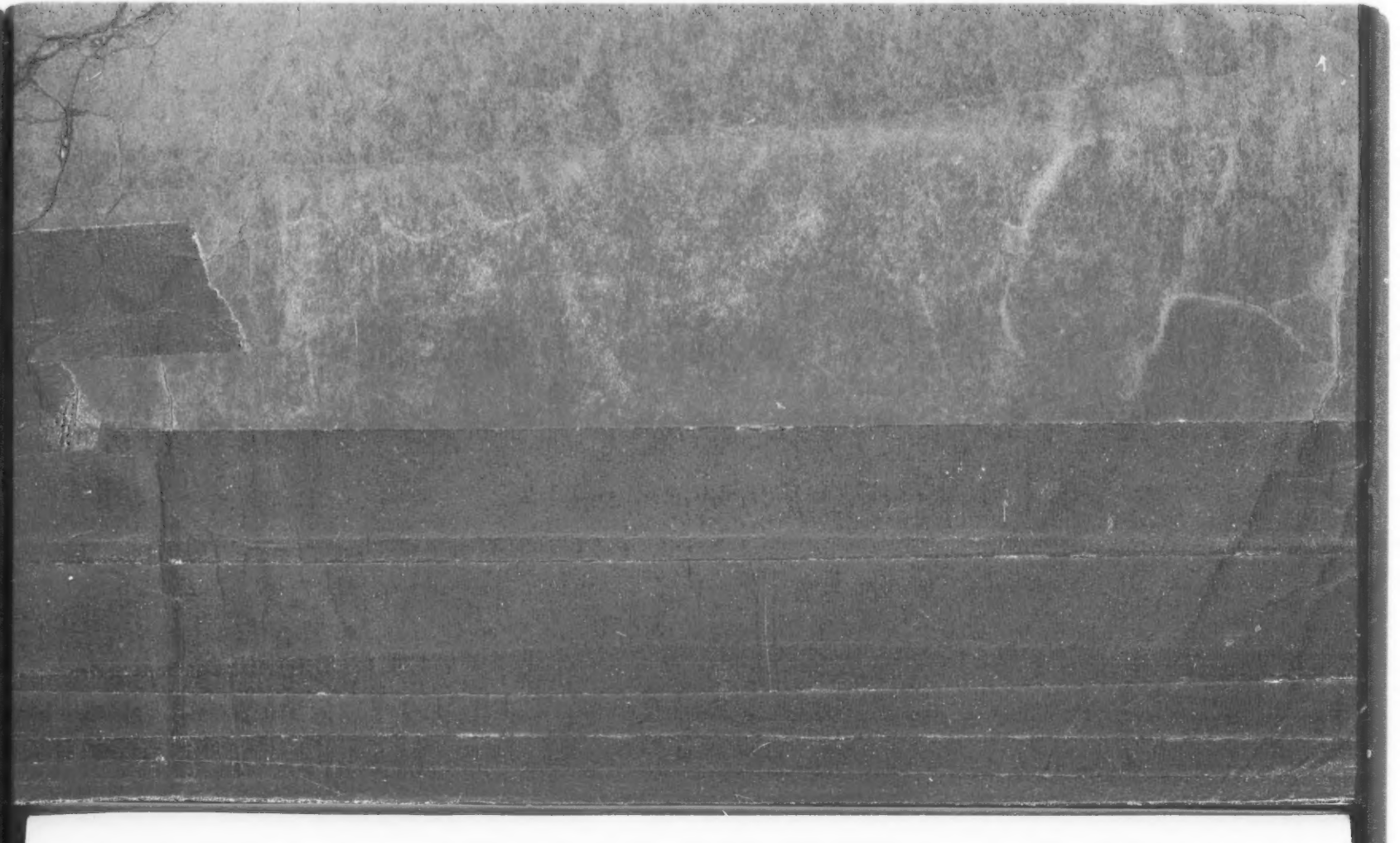
REVIEWS:

BARNETT, S. A. *et al.* A century of Darwin . . 521

A handbook on evolution . . . . . 521

SHEPPARD, P. M. Natural selection and heredity . 522

HUTCHINSON, SIR JOSEPH. Genetics and the improvement of tropical crops . . . . . 523

GENETICAL SOCIETY OF GREAT BRITAIN: Abstracts of Papers . . . . . 525

INDEX . . . . . 529